A STUDY ON eGFR, PROTEIN-CREATININE RATIO, THYROID AND LIPID PROFILE ON ASSESSING RISK FACTOR FOR RENAL DYSFUNCTION

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY,
CHENNAI – 600032.

In partial fulfillment of the regulations
For the award of the degree
M.D. BIOCHEMISTRY BRANCH-XIII

DEPARTMENT OF BIOCHEMISTRY

TRICHY SRM MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE
(Formerly Chennai Medical College Hospital and Research Centre)
IRUNGALUR,TRICHY – 621 105.

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY,
CHENNAI – 600032

MAY 2019

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON eGFR, PROTEIN-CREATININE RATIO, THYROID AND LIPID PROFILE ON ASSESSING RISK FACTOR FOR RENAL DYSFUNCTION"** is a bonafide work done by **Dr.T.JAYAKALA,** in partial fulfillment of the requirements for M.D., Branch –XIII (Biochemistry) examination of **The Tamil Nadu Dr. M.G.R Medical University,** Chennai to be held in May – 2019. The period of study was from 2016-2019.

Dr.A. JESUDOSS, M.S., DLO.,

The Dean,

Trichy SRM Medical College

Hospital & Research Centre,

Irungalur,Trichy-621105

Dr. KALAVATHY PONNIRAIVAN,M.D.,

Professor and Head,

Department of Biochemistry,

Trichy SRM Medical College

Hospital &Research Center,

Irungalur,Trichy-621105

GUIDE CERTIFICATE

GUIDE : **Prof. Dr. KALAVATHY PONNIRAIVAN M.D.,**
Professor and Head of the Department,
Department of Biochemistry,
Trichy SRM Medical College Hospital and Research Centre,
Irungalur, Trichy-621105.

CO-GUIDE : **Dr.SP.S SUBRAHMANIAN M.D., DM(Nephrology)**
Assistant Professor,
Department of Nephrology
Trichy SRM Medical College Hospital and Research Centre,
Irungalur, Trichy-621105.

Remark of the Guide:

This work by **Dr.T.JAYAKALA**, on **"A STUDY ON eGFR, PROTEIN-CREATININE RATIO, THYROID AND LIPID PROFILE ON ASSESSING RISK FACTOR FOR RENAL DYSFUNCTION"** was done under my supervision and I assure that this candidate has abided by the rules of the Ethical committee.

Guide

Prof. Dr. KALAVATHY PONNIRAIVAN M.D.,
Professor and Head of the Department,
Department of Biochemistry,
Trichy SRM Medical College Hospital and
Research Centre, Irungalur, Trichy-621105.

DECLARATION BY THE CANDITATE

I, **Dr.T. Jayakala,** here by solemnly declare that the dissertation titled **"A STUDY ON eGFR, PROTEIN-CREATININE RATIO, THYROID AND LIPID PROFILE ON ASSESSING RISK FACTOR FOR RENAL DYSFUNCTION"** was done by me at Trichy SRM Medical College Hospital and Research Centre, Irungalur, Trichy under the supervision and guidance of Professor and Head of the Department **Dr.Kalavathy Ponniraivan, M.D.,** This dissertation is submitted to the TamilNadu Dr. M.G.R. Medical University, Chennai, towards partial fulfillment required for the award of M.D., Degree (Branch XIII) in Biochemistry.

Place: Irungalur, Trichy

Date:

Signature of the Candidate

(Dr.T.JAYAKALA)

INSTITUITIONAL ETHICS COMMITTEE CERTIFICATE

CHENNAI MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE

IRUNGALUR, TRICHY – 621 105.

E.Mail : researchcmchrc@gmail.com, Phone: 0431-3058863,3058817

INSTITUTIONAL ETHICS COMMITTEE CERTIFICATE.

Ref.No: CMCH&RC/IEC –**No 31- :11.04.2017**

Sub: Approval of research work / project of faculty- IEC – Issued-Reg.

The research proposal submitted by **Dr. T. Jayakala-** I year post graduate, Department of Biochemistry, Chennai Medical College Hospital & Research Centre, was discussed by the Institutional Ethics Committee of the CMCH&RC. The committee approved the research project subject to existing rules and regulations.

Title of the Research work/Project: A study on eGFR, protein – creatinine ratio, Thyroid and lipid profile on assessing risk factor for renal dysfunction.

a. She should abide to the Ethical aspects of the institutional Ethics Committee.

b. She should not deviate from the proposal submitted

c. She has to inform IEC if any deviation / modification whenever considered.

d. She should carry out the project within the stipulated period and if extension needed, She has to inform IEC

e .She should get appropriately designed informed consent subjects/patients of the study group.

f. She should not claim any monetary support from IEC

g. She should cooperate with the members of IEC while they visit / monitor the activities

h. She should entitle to make use of this approval letter for obtaining financial support from funding agencies by submitting his application through the Dean, CMCH&RC.

i. She is informed that she should submit the summary of the report to the IEC after completion of her project

k. **IEC Comments: Approved, Consent from study subjects to be obtained** and **kept in her custody for verification by IEC any time.**

Member Secretary
[DR.S.D. Nalinakumari]

TO

Dr. T. Jayakala , I year Post Graduate, Department of Biochemistry, CMCH&RC, Trichy.

Copy to office

INSTITUTIONAL ETHICS COMMITTEE * Chennai Medical College Hospital & Research Centre Irungalur, Trichy-621 105 Date

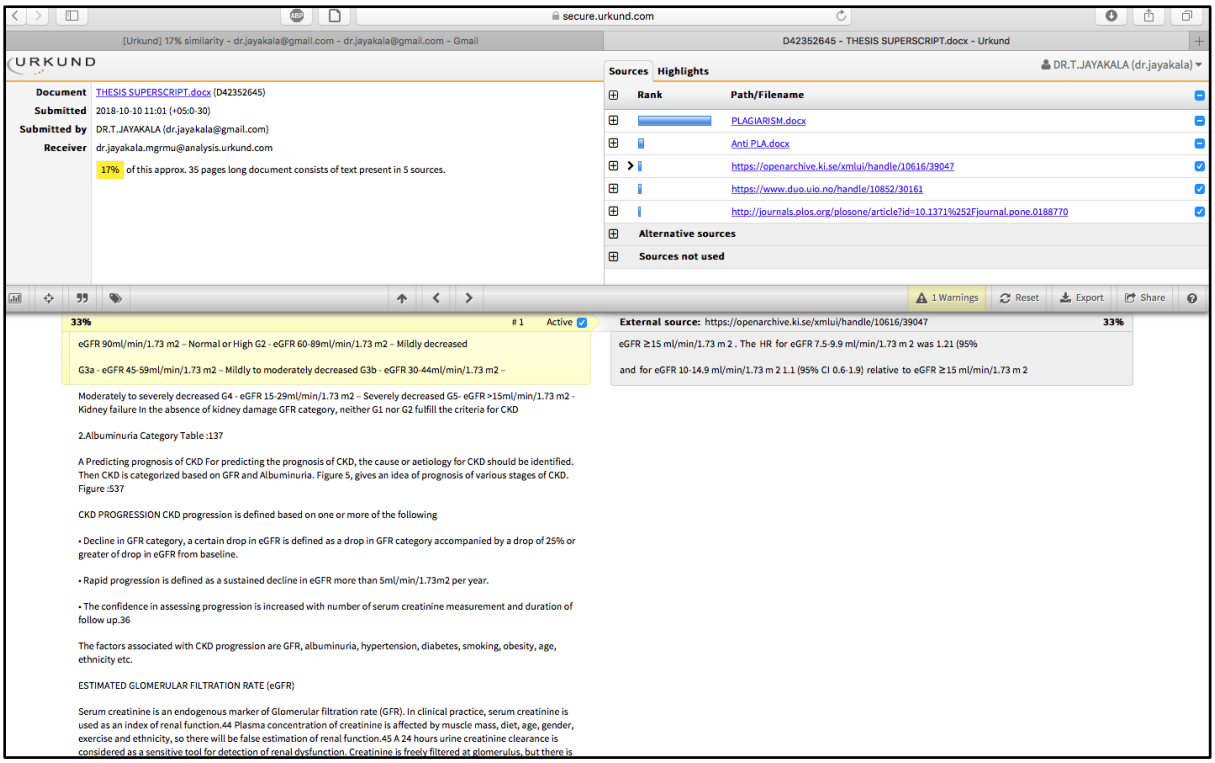
CERTIFICATE II

This is to certify that this dissertation work titled **"A STUDY ON eGFR,PROTEIN-CREATININE RATIO, THYROID AND LIPID PROFILE ON ASSESSING RISK FACTOR FOR RENAL DYSFUNCTION"** of the candidate **Dr.T.JAYAKALA** with Registration Number **201623401** for the award of **M.D. DEGREE** in the Branch–**XIII BIOCHEMISTRY**. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **17%** plagiarism in the dissertation.

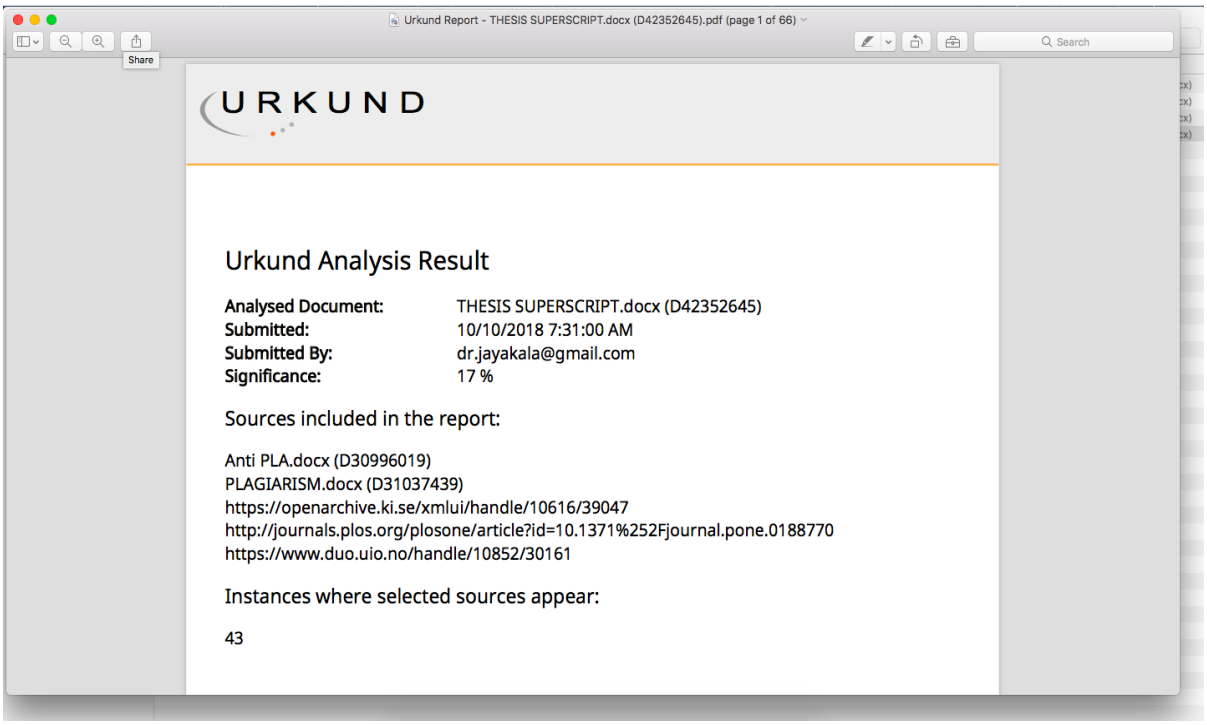
Guide & Supervisor

Prof. Dr. KALAVATHY PONNIRAIVAN M.D.,
Professor and Head of the Department,
Department of Biochemistry,
Trichy SRM Medical College Hospital and
Research Centre, Irungalur, Trichy-621105.

ANTI-PLAGIARISM- ORIGINALITY REPORT



PLAGIARISM-ANALYSIS REPORT



ACKNOWLEDGEMENT

I am thankful to the Chairman Dr.R.Shivakumar, M.D., and the Dean Dr.A.Jesudoss M.S.,DLO., of Trichy SRM Medical College Hospital and Research Centre, Irungalur for giving permission to do the research work in our hospital.

My sincere gratitude to my guide Prof. **Dr. KalavathyPonniraivan M.D.**, Professor and Head of the Department, Department of Biochemistry, Trichy SRM Medical College Hospital and Research Centre, for her valuable guidance, novel ideas, constant support and encouragement since the beginning of the study.

I am thankful to my Co-Guide Dr.SP.S.Subrahmanian M.D., DM(Nephrology), Assistant Professor, Department of Nephrology, Trichy SRM Medical College Hospital and Research Centre, Irungalur, for helping me out in the study whenever needed, besides his busy schedule.

I am thankful to Prof. Dr.H.Geetha PhD, for her support during the study period.

I am grateful to Dr.R.Thamarai M.D., Associate Professor, Department of Biochemistry, Trichy SRM Medical College Hospital and Research Centre, for her constructive suggestions and opinions during the study and helping me in bringing up the work in a complete form.

I am thankful to Dr.A.Velayutharaj M.D., Assistant Professor Department of Biochemistry, Trichy SRM Medical College Hospital and Research Centre, for his support.

I am thankful to Dr.M.Rasheedhkhan M.D.,and Dr.T.M.Moonishaa M.D., Assistant Professors Department of Biochemistry, Trichy SRM Medical College Hospital and Research Centre for their timely advises, tips and encouragement during the study period.

I am thankful to Dr.K.Hemalatha M.D., Associate Professor, Department of Community Medicine,Trichy SRM Medical College Hospital and Research Centre, Irungalur. She has been patient enough to help me out in Statistical analysis of the study despite her tight schedule.

I am thankful to Dr.K.Ilavenil M.Sc., Tutor Department of Anatomy for the help rendered.

My sincere thanks to Dr.Arya M.D., Dr.P.Sivakumar M.D., Dr.P.Lawrence M.D., Dr.R.Sowrirajan M.D., and Dr.M.PraveenKumar M.D**.,** Assistant professors of Department of Medicine, for helping me to identify patients in OPD from the very beginning of my study.

I am thankful to my friends Dr.D.BellaDevaleenal, Dr.S.Kalavathy, Dr.S.Santhini and Dr.Freethi for their suggestions and for helping me in the dissertation.I am thankful to all the laboratory staffs of Biochemistry and Pathology, and the outpatient department staffs who helped me during the study.

I am indebted to my son J.NaveenAdithya for his patience and for encouraging me during my tough days. I thank my family members for their moral support.

Above all I thank GOD for his blessings throughout the study period.

CONTENTS

ABBREVATIONS

AKI- Acute Kidney Injury

AKD- Acute Kidney Disease

AA- Afferent Arteriole

ADMA- Asymmetric Di Methyl Arginine

ADH- Anti Diuretic Hormone

ANP – Atrial Natriuretic Peptide

AER- Albumin Excretion Rate

ACR – Albumin Creatinine Ratio

AGE- Advanced Glycated End product

AchR- Acetyl Choline Receptor

BMI- Body Mass Index

BNP-B- B type Natriuretic Peptide-B

BP- Blood Pressure

CVD- Cardio Vascular Disease

CKD- Chronic Kidney Disease

CKD- EPI – Chronic Kidney Disease Epidemiology Collaboration Group

CHE- Cholesteryl Esterase

CHO- Cholesterol Oxidase

CRP- C-Reactive Protein

DM- Diabetes Mellitus

DNA- Deoxy Ribonucleic Acid

EA- Efferent Arteriole

EPO- Erythropoietin

EC- Extracellular

ECLIA- Electro ChemiluminescenceImmuno Assay

eGFR- Estimated Glomerular Filtration Rate

ESRD- End Stage Renal Disease

FA- Fatty Acid

FT3 – Free Triiodothyronine

FT4- Free Thyroxine

GAPDH- Glyceraldehyde-3-phosphate dehydrogenase

GLDH- Glutamate dehydrogenase

GOD- Glucose Oxidase

GLUT-Glucose Transporter

HT- Hypertension

HTN- Hypertension

HDLc- High Density Cholesterol

HGF- Hepatocyte Growth factor

IGF-1- Insulin like Growth Factor-1

IL-6- Interleukine -6

KIM-1-Kidney Injury Marker-1

K-DOQI- National Kidney Foundation Disease Outcomes Quality Initiative

KDIGO- Kidney Diseases Improving Global Outcome

LDLc- Low Density Lipoprotein Cholesterol

MeN- Mesoamerican Nephropathy

MDRD- Modified Diet In Renal Diseases

MAPK-Mitogen Activated Protein Kinase

NAP- Non Albumin Proteinuria

N-GAL-Neutrophil Gelatinase Associated Lipocalin

NSAID-Non Steroidal Anti Inflammatory Drug

NO- Nitric Oxide

Na- Sodium

NKCC2- (NaK2Cl)- Sodium Potassium Chloride pump

OSAS- Obstructive Sleep Apnoea Syndrome

PCT – Proximal Convoluted Tubule

PDGF- Platelet Derived Growth Factor

PCR- Protein Creatinine Ratio

PKC- Phospho Kinase C

PARP- Poly-Adenosine di phosphate-Ribose Polymerase

RAS- Renin Angiotensin System

RAAS Renin Angiotensin Aldosterone System

ROS- Reactive Oxygen Species

SREBP- Sterol Regulatory Element Binding Protein

SEEK- Screening and Early Evaluation of Kidney Disease

TC- Total Cholesterol

TSH- Thyroid Stimulating Hormone

TGL- Triglycerides

TNF-∞- Tumor Necrosis Factor alpha

TZD- Thiazolidinediones

UPCR – Urinary Protein Creatinine Ratio

VEGF- Vascular Endothelial Growth Factor

VSMC-Vascular Smooth Muscle Cell

WC- Waist Circumference

INTRODUCTION

Chronic Kidney Disease (CKD), is a major health problem in India. As per the Kidney Diseases Improving Global Outcomes (KDIGO) 2012, CKD is defined[1] as abnormalities of kidney structure or function present for more than three months. Presence of either of the following markers of kidney damage (one or more) present for > 3 months is considered as criteria for CKD.

1. Decreased Glomerular Filtration Rate (GFR) < 60 ml/min/1.73 m^2.

2. Albuminuria (Albumin Excretion Rate (AER) ≥ 30 mg/ 24 hrs, Albumin Creatinine Ratio (ACR) ≥ 30 mg/g).

3. Urine Sediment abnormalities.

4. Electrolyte and other abnormalities due to tubular disorder.

5. Abnormalities detected by histology.

6. Structural abnormalities detected by imaging.

7. History of Kidney transplantation.

As per the Screening and Early Evaluation of Kidney Disease (SEEK) India, study the prevalence of CKD in India is 17.2%(Stage1-5).[2] The prevalence of CKD globally remain up to 13.4%.[3]As per KDIGO 2012, CKD stages are determined according to estimated Glomerular Filtration Rate (eGFR) categories as G1-G5 and Albuminuria Categories as A1-A3, stressing the importance of albuminuria in predicting the prognosis of CKD.[1]

An upsurge in CKD has been attributed to the following risk factors age, sex, race, hypertension, diabetes mellitus, obesity, smoking, cardiovascular disease, exposure to nephrotoxic drugs, agrochemicals, herbal medication and food additives.[4,5] Serum elevation of excretory products like metabolites of purines, amino acid catabolism (urea and uric acid) has been attributed to decreased GFR.[6,7,8]

There is growing evidence that abnormalities in lipid metabolism may also contribute to progression of renal dysfunction.[9] High level of Triglycerides (TGL) and low High Density Cholesterol (HDLc) are identified as risk factors of renal dysfunction.[10,11] The ratio of TGL/HDLc and renal dysfunction is under study and a study among adult Koreans has stated that elevated TG/HDLc ratio is associated with renal dysfunction.[12] Injury induced by cellular TGL accumulation can cause disruption and damage to the cellular homeostasis. TGL accumulation caused renal tubular damage in both, in vivo and in vitro models.[13] HDLc promote TGL clearance from circulation and prevents lipid deposition in the arterial walls which may slow the progression of CKD.[14,15]

Thyroid hormones were also found to influence GFR, tubular secretion and absorption.[16,17]One study has shown that normal to high levels of Thyroid Stimulating Hormone (TSH) and normal to low levels of free triiodothyronine (FT3) were associated with increased risk of CKD in euthyroid subjects.[18] It is also said that high level of serum free thyroxine (FT4) was associated with increased risk of CKD rather than TSH and FT3.There was also associated rapid decline in eGFR.[19]Many studies have found that hypothyroidism is associated with dyslipidemia and renal dysfunction.[20,21,22] The association of thyroid dysfunction and dyslipidemia may play an important role in pathogenesis of renal dysfunction.Heat stress and dehydration are considered as risk factors for CKD. The pathophysiology is due to dehydration, decreased renal blood flow and increased level of uric acid.[23]

Agrochemicals like 2,4 D and paraquat dichlorate, non-steroidal anti-inflammatory drugs(NSAID), heavy metal exposure are associated with acute kidney injury.[23,24,25] Studies suggest that in obesity, adipose tissue may contribute to renal damage by oxidative stress, abnormal plasma lipid, coagulation abnormalities,

endothelial dysfunction, inflammation and insulin resistance.[26,27] Smoking is associated with CKD, the possible mechanism includes increased blood pressure and heart rate, cell proliferation, fibronectin, arteriosclerosis of renal artery and arterioles, increased production of growth factors like angiotensin II, endothelin-I, transforming Growth Factor-1, oxidative stress, tubular toxicity, increased aggregation of platelet and vasopressin mediated antidiuresis.[28,29]

There is not much of study in India to assess the risk factors and early detection of renal dysfunction in a heterogeneous group of population. Community based screening programs for early detection of renal dysfunction will prevent progression to CKD and decreases morbidity and mortality. Identification of novel risk factors can help in early detection of renal dysfunction.

AIM OF THE STUDY

To assess the risk factors for renal dysfunction in a heterogeneous population.

OBJECTIVES OF THE STUDY

1. To determine the age and sex wise distribution of eGFR and Urinary spot Protein- Creatinine ratio (UPCR) in a heterogeneous population.
2. To find out prevalence of various CKD stages based on eGFR .
3. To estimate the lipid profile and thyroid profile and assess their role as risk factor in renal dysfunction.
4. To find out the association between lipid profile and thyroid profile and different stages of CKD.
5. To find out the association between the risk factors like Obesity, Diabetes mellitus, Hypertension and Cardiovascular disease and different stages of CKD.

REVIEW OF LITERATURE

KIDNEY

The kidney is a functionally diverse organ and participates in many of the body's homeostatic mechanisms. Kidneys are a pair of bean shaped organs located in the retroperitoneal space. Each kidney is divided into an outer cortex and inner medulla. The Nephron is the functional unit of kidney. Each kidney consists of about a million nephrons. Each nephron consists of

1. Glomerulus, which is a tuft of capillaries surrounded by expanded end of renal tubule called Bowman's capsule, supplied by an afferent and efferent arteriole.
2. Proximal Convoluted tubules, which are tightly coiled in the beginning called as pars convoluta, which towards the medulla become straightened to form pars recta.
3. Loop of Henle, consists of the thin descending limb and the thick ascending limb.
4. Distal convoluted tubule.
5. Collecting duct, formed by two or more distal convoluted tubules that drains urine from each nephron. Collecting ducts merge and empty their content into the renal pelvis.[30]

Figure: 1shows the anatomy and organization of kidney.

Figure: 2 and 3 show the structure of nephron.

Figure1[30]: Organization of kidney[30]

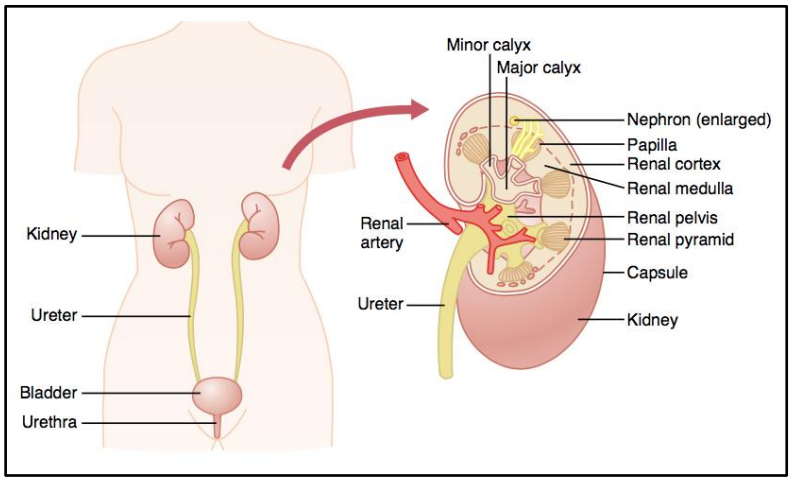


Figure2[30]: Basic Tubular Segments of aNephron[30]

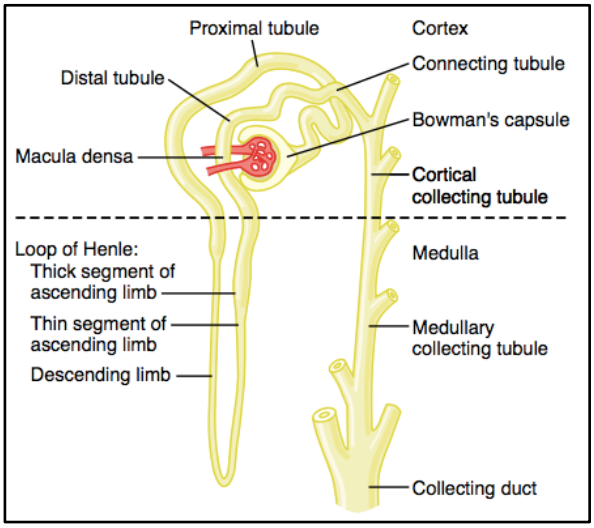
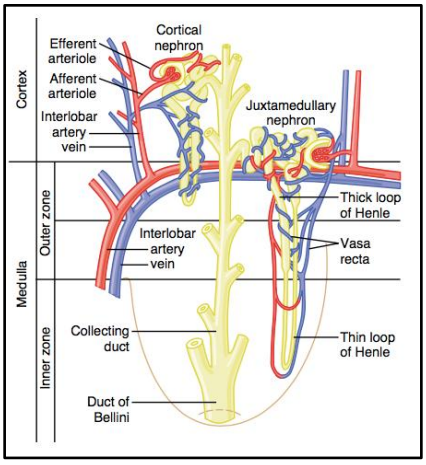


Figure 3[30]:Cortical and Juxtamedullary Nephrons[30]



KIDNEY FUNCTION

The basic functions of kidney are[30]

1. Urine formation
2. Fluid and electrolyte balance
3. Regulation of acid base balance
4. Excretion of waste products of metabolism
5. Secretion of hormones
    - Renin
    - Erythropoietin
    - 1,25, dihydroxy Vitamin D_3 (Calcitriol)
    - Prostaglandin

KIDNEY DISEASES

The kidney diseases have been classified as

1. Acute Kidney Disease (AKD).
2. Chronic Kidney Disease (CKD).
3. End Stage Renal Disease (ESRD).

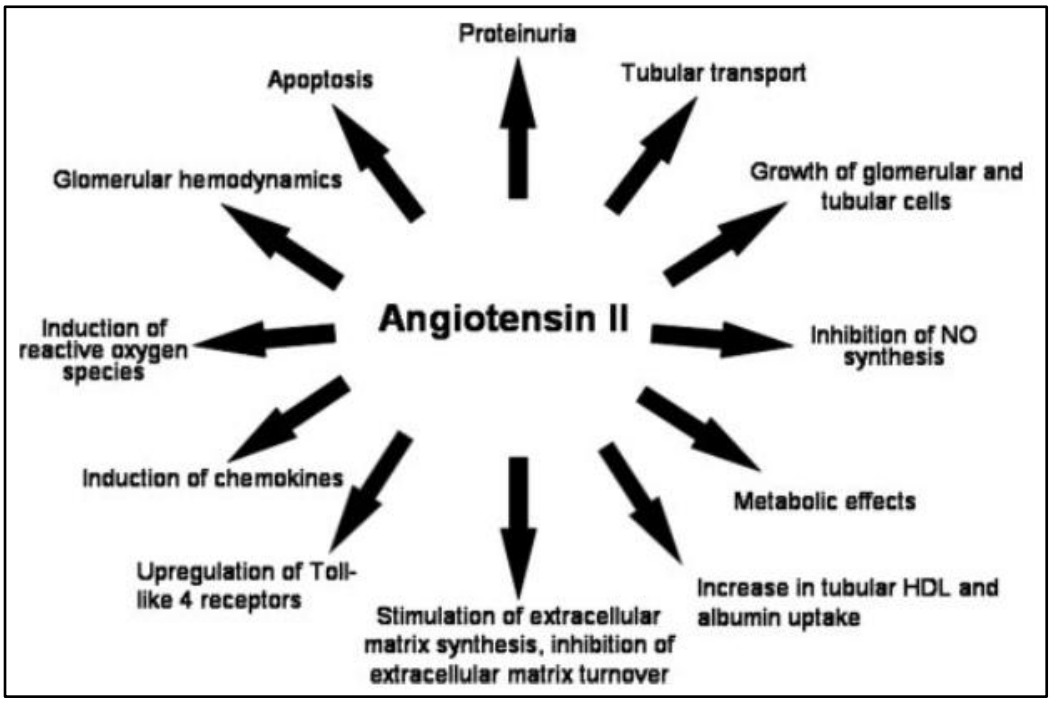
PATHOPHYSIOLOGY OF KIDNEY DISEASES

Irrespective of the etiology of the kidney disease, the pathogenesis leading to renal dysfunction remain the same. The pathological process is characterized by,early inflammation followed by accumulation and deposition of extracellular matrix, leading to tubulointerstitial fibrosis andtubular atrophy and finally leading to glomerulosclerosis.[31]

The local Renin Angiotensin Aldosterone System (RAAS) has an important role in the pathological process of kidney disease.[32] Angiotensin II produces toxic reactive oxygen species (ROS) within the renal cells and affect signal transduction. It also induces many inflammatory mediators like cytokines, chemokines and growth factors which activates the lymphocytes and macrophages. The activated cells further causes lysis of cells and proliferation of interstitial fibroblast which causes increase in the extracellular matrix and fibrosis of the glomerulus and tubules. Blood supply will be affected leading to cell death.[31,32] The figure 4,gives an outline of the pathological process causing renal disease.

Figure:4[32]

Role of Angiotensin II in renal pathology



The kidneys increase their functional capacity after injury. Symptoms or major biochemical changes occur occur only if 50-60% of the functioning kidney mass is lost.There will be increase in workload of the nephron and is considered as an important cause of progressive renal injury.[31,33] A hypothesis is that a point at which

the decline in nephron number is reached, further loss in nephron will lead to progressive renal disease as a consequence of a common pathway leading to interstitial fibrosis.[31,34]

CHRONIC KIDNEY DISEASE

The increasing incidence of lifestyle disorders may cause resource crunch in economy of our country. One such disease which India faces is Chronic Kidney Diseases. CKD constitute a major cost burden in health care system worldwide. The incidence of CKD is increasing worldwide. As per the Screening and Early Evaluation of Kidney Disease (SEEK) India,study the prevalence of CKD in India is 17.2%.[2] The prevalence of CKD globallyremains up to 13.4%.[3]

Definition of CKD[35,36,37]

According to Kidney Diseases Improving Global Outcomes (KDIGO) 2012, Chronic Kidney Disease(CKD) is defined as abnormalities of kidney structure or function present for more than three months.

Criteria of CKD (either of the following present for > 3 months)

1. Markers of Kidney damage (one or more)[35,36,37]

- Albuminuria (AER ≥ 30 mg/24 hours, ACR ≥ 30 mg/g / ≥ 30 mg/mmol)
- Structural abnormalities detected by imaging.
- Urine Sediment abnormalities.
- Electrolyte and other abnormalities due to tubular disorders.
- Abnormalities detected by histology.
- History of Kidney transplantation.

2. Decreased GFR[35,36,37]

- GFR <60 ml/min/1.73 m^2 (GFR categories G3a-G5)

- The criteria GFR <60 ml/min/1.73m^2 for CKD is selected because at this value approximately one half of adult renal function is lost.[38,39]

RISK FACTORS OF CKD

Risk factors which are associated with renal dysfunction include hypertension(HT), diabetes mellitus (DM), cardiovascular disease (CVD), proteinuria, anaemia.[40]The non-traditional CKD risk factors include nephrotoxin exposure, renal stones, acute kidney injury (AKI), infections,[41] dyslipidemia are associated with renal disease progression.[42]Thyroid dysfunction is also a risk factor for progression of renal dysfunction.[43]

GRADING OF CKD[35,36,37]

As per KDIGO 2012, CKD is classified based on

1. GFR category and

2. Albuminuria category

1.GFR Category [37]

G1 - eGFR ≥ 90ml/min/1.73 m^2 – **Normal or High**

G2 - eGFR 60-89ml/min/1.73 m^2 – **Mildly decreased**

G3$_a$ - eGFR 45-59ml/min/1.73 m^2 – **Mildly to moderately decrea**sed

G3$_b$ - eGFR 30-44ml/min/1.73 m^2 – **Moderately to severely decreased**

G4 - eGFR 15-29ml/min/1.73 m^2 – **Severely decreased**

G5- eGFR <15ml/min/1.73 m^2 **- Kidney failure**

In the absence of kidney damage GFR category, neither G1 nor G2 fulfill the criteria for CKD

2. Albuminuria Category

Table :1[37]

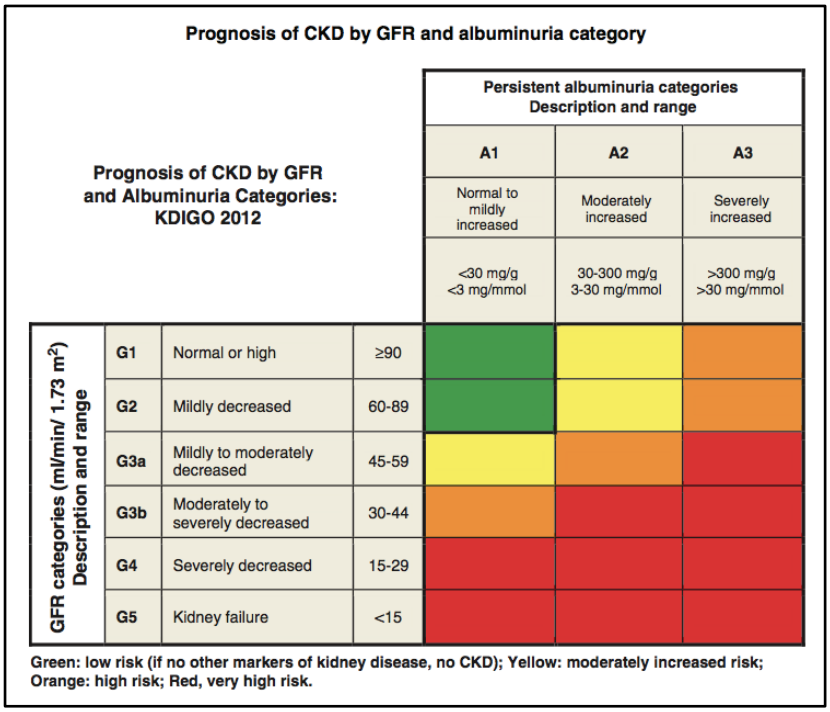
Category	AER (mg/24 h)	ACR (approximate equivalent) (mg/mmol)	ACR (approximate equivalent) (mg/g)	Terms
A1	<30	<3	<30	Normal to mildly increased
A2	30–300	3–30	30–300	Moderately increased*
A3	>300	>30	>300	Severely increased**

Abbreviations: ACR, albumin-to-creatinine ratio; AER, albumin excretion rate; CKD, chronic kidney disease.
*Relative to young adult level.
**Including nephrotic syndrome (albumin excretion usually >2200 mg/24 h (ACR >2220 mg/g; >220 mg/mmol)).

Predicting prognosis of CKD

For predicting the prognosis of CKD, the cause or aetiology for CKD should be identified. Then CKD is categorized based on GFR and Albuminuria. Figure 5, gives an idea of prognosis of various stages of CKD.

Figure 5:[37]



CKD PROGRESSION

CKD progression is defined based on one or more of the following

- Decline in GFR category, a certain drop in eGFR is defined as a drop in GFR category accompanied by a drop of 25% or greater of drop in eGFR from baseline.
- Rapid progression is defined as a sustained decline in eGFR more than 5ml/min/1.73m^2 per year.
- The confidence in assessing progression is increased with number of serum creatinine measurement and duration of follow up.[36]

The factors associated with CKD progression are GFR, albuminuria, hypertension, diabetes, smoking, obesity, age, ethnicity etc.

ESTIMATED GLOMERULAR FILTRATION RATE (eGFR)

Serum creatinine is an endogenous marker of Glomerular filtration rate (GFR). In clinical practice, serum creatinine is used as an index of renal function.[44] Plasma concentration of creatinine is affected by muscle mass, diet, age, gender, exercise and ethnicity, so there will be false estimation of renal function.[45] A 24 hours urine creatinine clearance is considered as a sensitive tool for detection of renal dysfunction. Creatinine is freely filtered at glomerulus, but there is some tubular secretion. Also the timed urine collection is difficult. There was a wide(11%) within subject variability. These factors affected the urine clearance method of GFR estimation.[44]

The National Kidney Foundation Disease Outcomes Quality Initiative (K-DOQI) recommended the use of estimated GFR (eGFR)calculated by using formulas based on plasma or serum creatinine.[36] Cockcroft and Gault in 1976 published an

equation based on age, weight, height and plasma creatinine with correction factors.[46]The limitation of this formula is that, it was derived from hospitalised individuals mostly men. Also the need for height and weight measurement for calculation is a limitation.[45]

A Modification of Diet in Renal Disease (MDRD) study, evaluated the effect of protein restricted diet on renal disease progression and created an equation for calculation of eGFR, which included age, gender, plasma creatinine and race differentiation (white or black).[47]MDRD did not require body weight or height. As MDRD was originally derived from a group of CKD patients, its use in healthy individuals is unclear. It is not validated for children, pregnant women, obesity and elderly patients (>70 years).[45] At a low creatinine value the performance of MDRD formula was poor.[38,48]

Chronic Kidney Diseases-Epidemiology Collaboration group (CKD-EPI) developed a new formula in 2009, to have greater accuracy even at high GFR. It includes race, gender, and age.[49] Many studies have found cystatin C derived equation for eGFR is better than creatinine in predicting renal function.[50,51] Schwartz formula developed in 1970 is used to estimate GFR in chidren.

Schwartz Formula for children[52]

eGFR=0.41x height (cm)/ S_{cr} (mg/dl) (updated Schwartz bedside formula)

The best equation for GFR in children was [52,53]

eGFR=39.1X [height(m^2)/ S_{cr} (mg/dl)]$^{0.516}$ X [1.8/cystatin C (mg/L)]$^{0.294}$X [30/BUN(mg/dl)] X[1.099^{male}] X [height(m)/1.4] $^{0.188}$

Equations for calculating eGFR:[45]

Table 2:[45]

MDRD eGFR = 186 x [Plasma Creatinine (µmol/L) x 0.0011312] $^{-1.154}$ x [age (years)] $^{-0.203}$ x [0.742 if female] x [1.212 if black] [equation 1]
MDRD eGFR (IDMS aligned) = 175 x [Plasma Creatinine (µmol/L) x 0.0011312] $^{-1.154}$ x [age (years)] $^{-0.203}$ x [0.742 if female] x [1.212 if black] [equation 2]
CKD-EPI eGFR; Female with Creatinine < 62 µmol/L; use eGFR = 144 x $(Cr/61.6)^{-0.329}$ x $(0.993)^{Age}$ Female with Creatinine > 62 µmol/L; use eGFR = 144 x $(Cr/61.6)^{-1.209}$ x $(0.993)^{Age}$ Male with Creatinine < 80 µmol/L; use eGFR = 141 x $(Cr/79.2)^{-0.411}$ x $(0.993)^{Age}$ Male with Creatinine > 80 µmol/L; use eGFR = 141 x $(Cr/79.2)^{-1.209}$ x $(0.993)^{Age}$ [equation(s) 3] where Cr is the plasma creatinine (µmol/L)

PROTEINURIA

Proteinuria is an independent risk factor for renal disease and cardiovascular disease.[54] The proteins of molecular weight of < 20,000 Da can be filtered by the glomerulus, the negative charge of the glomerular membrane restrict the passage of albumin through the membrane.[55] Normally the filtered protein are reabsorbed and catabolized in the proximal convoluted tubules. The normal protein excreted in urine is 150 mg/day. Excretion of albumin 30mg/day to 300mg/day is termed as microalbuminuria. Protein excretion >300mg/day is known as proteinuria or macro albuminuria.[56] Of the proteins excreted in urine, the high molecular weight albumin(65,000Da) is about 40%, low molecular weight like light chain immunoglobulin (20,000 Da) is of 20% and about 40% of Tamm-Horsfall mucoprotein (90,000 Da)[55].

Pathologic Proteinuria can be classified into three types

1. Glomerular proteinuria
2. Tubular proteinuria
3. Overflow proteinuria

Glomerular proteinuria is due to the permeability of glomerulus to high molecular weight proteins like albumin.[56]Glomerular proteinuria is the one which is used for staging of Chronic Kidney Disease. Measurement of albumin in urine determines the glomerular proteinuria.[55]

Tubular proteinuria results from decreased reabsorption of the protein present in the glomerular filtrate or due to the excretion of proteins produced due to tubular injury due to tubulointerstitial diseases like hypertensive nephrosclerosis, nephrotoxic drugs like nonsteroidal anti-inflammatory drugs etc.[55,56]

Overflow proteinuria is due to excess low molecular weight protein, which when excreted in normal amounts will be reabsorbed. Low molecular weight proteins that occur in overflow nephropathy are Bence–Jones protein of multiple myeloma, lysozyme in myelomonocytic leukemia, myoglobin in rhabdomyolysis.[55,56]

A 24–hour urine collection is considered as the gold standard in determining the urinary protein excretion. A 24-hour urinary protein collection show variation in urinary protein due to variation in intake of water, excretion of water, exercise, posture, diet etc. Nowadays expressing a spot urine protein as ratio to spot creatinine concentration is used. When the GFR remains constant the protein and creatinine excretion rates are constant during day[54].Currently spot urine protein- creatinine ratio (UPCR) is widely used to estimate the daily urinary protein excretion.[57] Microalbuminuria is an early sign of progression of renal and cardiovascular disease in patients who has hypertension and diabetes.[58] Proteinuria is a combination of

albuminuria and non albumin proteinuria(NAP). Isolated non albumin proteinuria (NAP) is a condition when urine total protein is elevated without elevation of urine albumin.[55] Non albumin proteinuria include alpha–microglobin, beta2 macroglobulin and IgG which are associated with renal damage in renal transplant patients.[59]

Non albumin proteinuria = Total protein – albuminuria.[60] Studies advice measurement of both urine spot albumin and total protein to identify the non albumin protein which may occur earlier than microalbuminuria.[55,60,61]

The KDIGO 2012 guidelines suggest early morning spot urine protein estimation in the following descending order,[37]

1. Urinary albumin –creatinine ratio (ACR).
2. Urine protein-creatinine ratio (PCR).
3. Reagent strip urinalysis for total protein with automated reading.
4. Reagent strip urinalysis for total protein with manual reading.

KDIGO also states that if non-albumin proteinuria is suspected, assays for specific urine proteins like Bence Jones protein is done.[37]

Consequences of proteinuria:

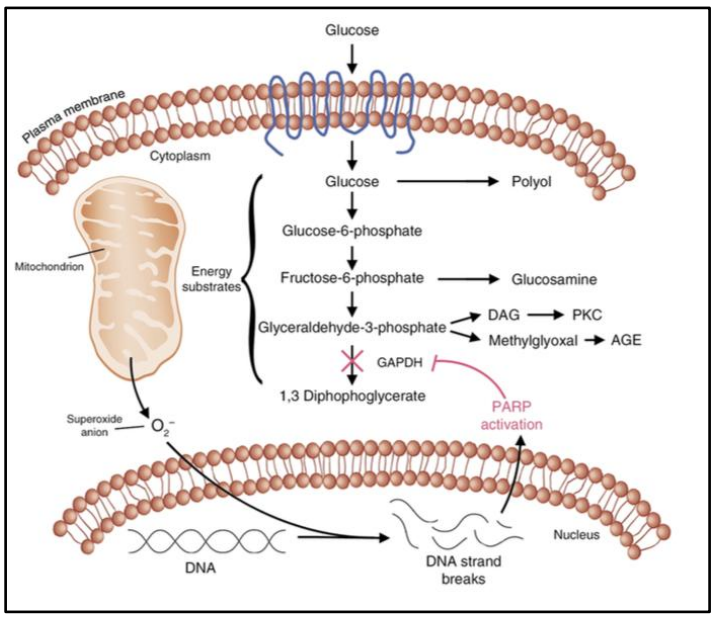
Proteinuria is not just a marker of renal dysfunction but it is also a risk factor for progression of kidney disease. The proteins accumulate in the tubular lumen causes inflammation and leads to interstitial structural damage and progression of kidney disease.[62] Also studies state that glomerular filtration of large amounts of protein by glomerulus causes mesangial cell injury and thus causing glomerulosclerosis[63] and progression of renal dysfunction.

DIABETES AND CHRONIC KIDNEY DISEASE

Diabetic nephropathy, one of the microvascular complications of diabetes mellitus, is one of the most important etiology for CKD and ESRD. Chronic hyperglycemia is the cause for pathogenesis of the microvascular complication.[64] The pathological finding in advanced diabetic nephropathy are glomerulosclerosis and tubulointerstitial fibrosis.[65] Experimental animal model studies have shown that in renal glomerulus and tubules there was increase in gene expression for synthesis of extracellular matrix proteins.[66] Hyperglycemia activate the polyol pathway of glucose metabolism and this leads to collagen accumulation in the renal tubular interstitium.[67,68,69,70] Hyperglycemia induces increased formation of advanced glycated end products of proteins,[71,72] growth factors/ cytokines like transforming growth factor-β (TGF-β),[73,74] platelet derived growth factors (PDGF),[75,76]insulin-like growth factor-I (IGF-I),[77,78] hepatocyte growth factor (HGF),[79] Vasoactive substances like angiotensin–II,[80,81] endothelin-I,[82] thromboxane,[83] which through autocrine and paracrine mechanism causes cell proliferation and growth.[84]

Figure 6, explains the mechanism of renal toxicity of hyperglycemia

Figure 6:[85]**Mechanism of Glucotoxicity on Kidney**



With increase in glucose, the mitochondria generate reactive oxygen species (ROS), which leads to break in DNA strands and activation of poly ADP ribose polymerase(PARP). PARP inactivates Glyceraldehyde 3-phosphate dehydrogenase(GAPDH) and the normal glucose metabolism is altered.

There will be

1. Activation of polyol pathway
2. Activation of Phosphokinase C(PKC) via diacyl glycerol.
3. Activation of Hexosaminase pathway and glucosamine gets accumulated.
4. Formation of Advanced glycated end products due to non enzymatic glycation of proteins.

All these mechanism produces increased growth factors and causes glomerular pathology.[64,85] Studies have shown that PKC stimulate the mitogen activated protein kinase (MAPK) pathway. The MAPK pathway, is an intracellular signal transduction pathway of cell growth, which activate cell growth and mesangial proliferation.[86,87] Further studies have proven that advanced glycated end products (AGE) also activate the MAPK pathway in proximal tubular LLC-PK_1 cells.[88]

Apoptosis causes the tubular atrophy and tubulointerstitial fibrosis.[89] Gene products like bcl2 is protective against apoptosis.[90] In a study there was increase in apoptotic cells in diabetic, db/db mice than in non-diabeticlittermates. Theapoptotic cells were found in the tubulointerstitium and not in glomeruli. There was increased expression of bax gene and a decrease in bcl-2 expression.[91]

DYSLIPIDEMIA OF CHRONIC KIDNEY DISEASE

Dyslipidemia is a common finding among CKD patients and is responsible for associated morbidity and mortality.[92] The plasma lipid profile in CKD shows elevated levels of triglycerides(TGL), low high density lipoprotein(HDLc),[93] increased very low density lipoprotein(VLDL), remnants and intermediate density lipoprotein,[94] accumulation of small dense low density lipoprotein(LDLc), glycated and carbamylated apolipoprotein,[95] prolonged persistent chylomicron remnant post prandially.[96] The composition of plasma lipoprotein is also altered in CKD.[97,98,99,100] In CKD the cholesterol content of VLDL is increased and there is relative decrease in TGL content. In LDLc the cholesterol content is decreased and there is relative increase in TGL content in CKD. In HDLc the cholesterol ester and free cholesterol content are reduced and TGL content is elevated.[54,55,56,57]There is hepatic lipase deficiency in humans and animals in CKD.[101] Normally hepatic lipase catalyses hydrolysis and removal of triglyceride content of HDLc. As there is deficiency of hepatic lipase there is increase in TGL content of HDL.[58] Elevated TG/HDL ratio can cause renal dysfunction.[102] The atherogenic lipoproteins like lipoprotein(a) and oxidized LDL are present in hemodialysis patients.[103]

Dyslipidemia causes renal damage and can cause progression of renal failure.[104] Dyslipidemia cause damage to mesangial cells, podocytes and glomerular capillary endothelial cells. Mesangial cells express LDL and oxidized LDL receptors, on activation there will be mesangial cell proliferation, increased mesangial matrix deposition, increase in cytokines like interleukin -6 or growth factors.Oxidized LDL increases the adhesion of monocytes to glomerular endothelial cells and causes monocyteinfiltration and causes tubular epithelial damage.[42,105] Hypercholesterolemia and hypertriglyceridemia causes podocyte injury and leads to mesangial sclerosis.[106]

In vivo studies have shown that there is increased expression of a transcription factor, sterol regulatory element binding protein(SREBP) which is associated with lipogenesis and increase in accumulation of lipid in the glomerulus and tubulointerstitial cell, leading to glomerulosclerosis and proteinuria.[107,108]

THYROID AND CKD

Thyroid hormones have its direct effect on kidney by causing systemic or local hemodynamic changes.[109]Thyroid hormone directly influence renal development and growth, GFR, renal transport systems and electrolyte homeostasis.[16,17,110]The presence of renal abnormalities in congenital hypothyroid children[111] indicate that thyroid hormone has a role in development of fetal kidneys. Animal studies have proved this.[112]

Effect of Thyroid Hormone on Renal physiology:

Thyroid hormone influences the renal function by action on cardiovascular system and there by the blood flow to the kidneys. Thyroid also affects the GFR, tubular secretion and reabsorption. In the proximal convoluted tubules, Na/K ATPase is influenced by thyroid hormones and thus the sodium reabsorption.[113] Thyroid hormone also influence the tubular potassium handling.[114] The renin angiotensin aldosterone axis is influenced by thyroid

hormone by regulating renin release,[115]adrenergic system[116]and angiotensinase activity.[117]Both hyperthyroidism and hypothyroidism has effects on kidney.

Role of Hyperthyroidism on Kidney function:

In hyperthyroid patients, an increase of 18-25% GFR has been noticed,[17] due to increased renal blood flow and renin angiotensin aldosterone system (RAAS) activation. There will be increased β-adrenergic activity in hyperthyroidism. Studies

have shown that thyroid hormone influence, increase in β-adrenergic receptors in renal cortex and is the cause for RAAS activation.[118] RAAS activity causes dilatation of afferent arteriole and constriction of efferent arteriole and increase filtration pressure leading to increase in GFR. Due to efferent arteriolar constriction, there will be decrease in the perfusion in proximal convoluted tubule (PCT) and there will be increased sodium and chloride reabsorption in PCT.[119]

Hyperthyroidism increases heartrate, raises cardiac output, decreases the systemic vascular resistance and thus increases systolic blood pressure.[120] Hyperthyroidism causes CKD by increasing the renal blood flow leading to intra glomerular hypertension and increased filtration pressure. Associated proteinuria in hyperthyroidism also causes renal injury.[121]

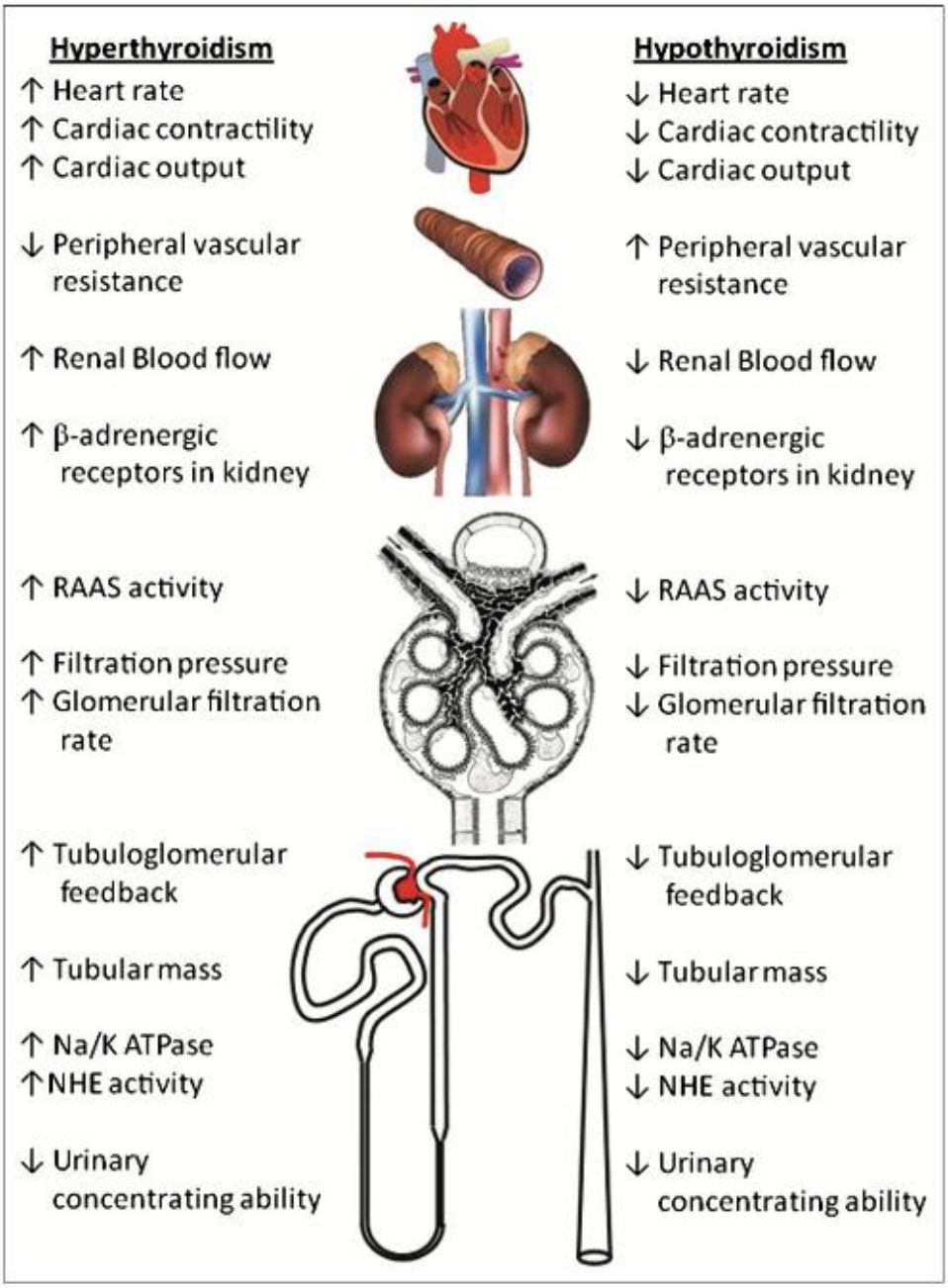
Role of Hypothyroidism on Kidney function

In adults and children, primary hypothyroidism is associated with a reversible increase in serum creatinine and decrease of GFR.[122,123,124,125,126,127] It has been reported that these defects were corrected with thyroid hormone replacement.[128] GFR measurement using Inulin or ^{51}Cr-EDTA has proved that hypothyroidism is associated with reduced GFR.[124] Hypothyroidism causes alteration in hemodynamics and structure of kidney.[129] The decrease in GFR in hypothyroidism is due to decreased renal blood flow.[130] Hypothyroidism also causes decreased cardiac output and low circulating volume. Decreased activity of renin angiotensin system(RAAS) and decreased levels of atrial natriuretic factor(ANF) are found in hypothyroidism and these cause decrease in renal perfusion.[109] Decreased expression of vascular endothelial growth factor(VEGF) and insulin like growth factor(IGF) which are renal vasodilators is seen in hypothyroidism.[131]

Figure7[121] gives an overview of effect of thyroid dysfunction on kidney.

Figure7 :[121]

Effect of Thyroid dysfunction on Kidney



Chronic Kidney Disease and Thyroid disease

Primary and subclinical hypothyroidism is common among CKD patients[132]. Decrease in T3(low T3 syndrome or euthyroid sick syndrome) is noticed in CKD patients.[133]This decrease in T3 (mainly total T3 and not free T3) is due to chronic protein malnutrition and metabolic acidosis affecting the iodothyronine deiodination

and decrease in T3 binding to protein.[134] Also decrease in deiodinase activity in CKD is due to inflammatory cytokines, which causes decrease in peripheral conversion of T4 toT3.[135]There is also decrease in thyroid hormone synthesis due to accumulation of inorganic iodine in CKD.[136]

OBESITY AND CKD

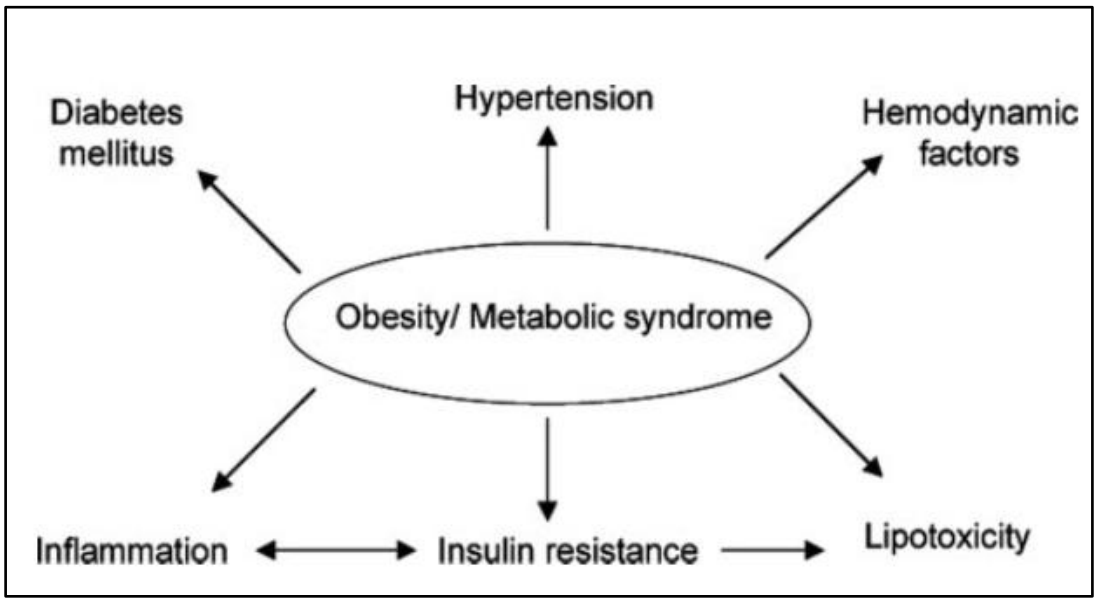
Obesity, overweight and metabolic syndrome are emerging as important independent risk factor for CKD.[137,138] Epidemiological studies have shown the association of obesity with CKD and end stage renal disease (ESRD). Studies showed that high BMI was associated with ESRD as an independent risk factor, independent of hypertension, proteinuria[139] smoking and diabetes.[140] Obesity was found toaffect the progression of preexisting renal diseases like IgA nephropathy,[141] unilateral renal agenesis[142] or after unilateral nephrectomy.[143] These studies suggest that obesity initiates CKD. A study of 6,500 nondiabetic subjects showed that increasing body mass index (BMI) and waist circumference(WC), had decreased eGFR and increased CKD.[27] Also obesity is closely associated with Type II diabetes and hypertension which are considered as important risk factors for CKD and ESRD.[144,145,146]

Mechanism of Obesity associated Kidney disease

Obesity causes renal damage directly by hemodynamic and hormonal effect[147,148] or indirectly by development of Type II diabetes mellitus and hypertension.[144,145,146]Figure 8,[149] gives an outline of mechanism of renal dysfunction in obesity.

Figure 8:[149]

Potential Mechanism of renal dysfunction in Obesity



Animal and human studies have shown that obesity causes increased renal tubular sodium reabsorption which causes renal vasodilatation[150] causing glomerular hyper perfusion and hyper filtration, which may cause focal segmental glomerulosclerosis and proteinuria.[148,151]The mechanism for early rise in GFR in obesity may be due to maculadensa feedback mechanism.[152] The visceral obesity causes compression of kidneys which may increase the sodium reabsorption in the loop of Henle[153] and hence less sodium in the macula densa and causes a tubulo glomerular feedback and increases renin secretion, increased blood flow and GFR. The glomerular hyper filtration then gradually subsides and there will be a gradual decrease in GFR with renal injury associated with prolonged obesity induced hypertension and diabetes. There will be increased glomerular wall tension and glomerular hypertrophy that leads to injury, glomerular sclerosis and nephron loss.[153,154] Activation of sympathetic nervous system(SNS)[155,156]and renin angiotensin aldosterone system (RAAS) also causes hypertension.[157]

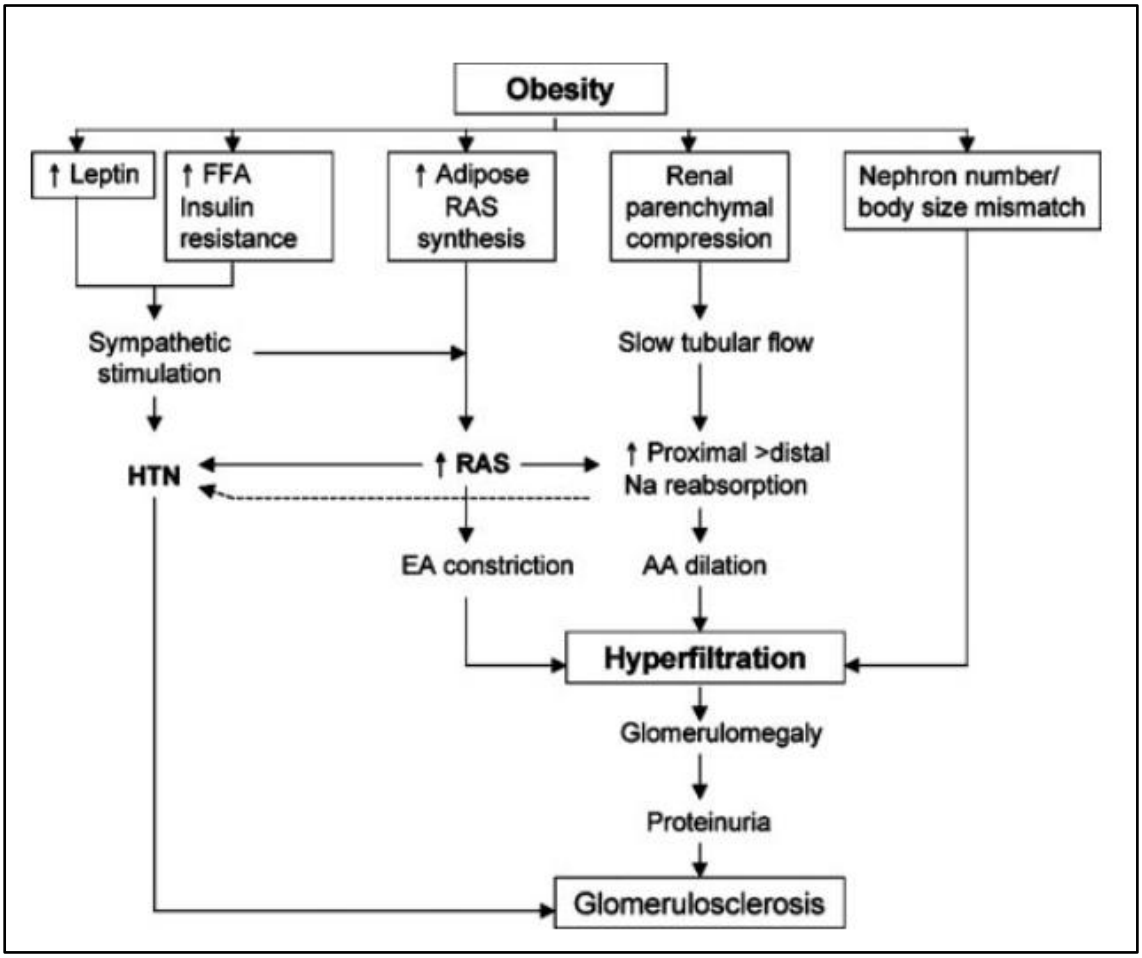
Obesity related glomerulopathy has been documented from kidney biopsy specimens.[148] Changes in the structure of kidney is noticed within few weeks of rapid weight gain. In animal experiments with dogs, enlargement of bowman's space, increase in glomerular cell proliferation, mesangial matrix and basement membrane thickening were observed. Glomerular transforming growth factor β, expression was also increased.[148]Also the leptin from adipose tissue might cause renal fibrosis.[157]Deposition of lipids in non adipose tissue like kidney leads to accumulation of diacylglycerol and ceramides, which may cause mitochondrial dysfunction, endoplasmic reticulum stress, apoptosis and renal injury and dysfunction.[158,159]

Inflammatory changes in Obesity causing CKD

Insulin resistance is present in metabolic syndrome, which is a proinflammatory state.[160,161]Visceral fat produce increased level of adipokines like interleukine-6 (IL-6), tumor necrosis factor, C-reactive protein (CRP) and resistin. A study has shown that in patients with obesity related glomerulopathy, the glomeruli showed increased expression of genes related to lipid metabolism, inflammatory cytokines and insulin resistance.[162] Leptin an adipocyte derived hormone, that suppresses appetite (hypothalamus) and increases insulin sensitivity. In obesity the hypothalamic effect of leptin is lost.[160,163,164]The leptin receptor (Ob-Ra) is expressed in kidney.[165] Recombinant leptin stimulate proliferation of cultured glomerular endothelial cells and also in rats.[166] Leptin stimulate type I collagen in cultured mesangial cells,[167] these show that leptin is involved in glomerulosclerosis. Figure 9,[149]shows an outline of mechanisms leading to renal dysfunction in obesity.

Figure 9:[149]

Hemodynamic consequences of Obesity leading to hyper filtration and hypertension



FFA- Free Fatty Acid;**RAS** –Renin Angiotensin System; **HTN**-Hypertension; **AA**-Afferent Arteriole; **EA** –Efferent Arteriole;**Na** –Sodium

CARDIOVASCULAR DISEASE AND CKD

Accelerated cardiovascular disease is a complication of renal disease. Mortality and morbidity due to cardiovascular disease is common in CKD patients. Studies have shown that prevalence of CKD is more among cardiovascular disease patients, CKD is also a risk factor for Cardiovascular disease (CVD),[168] and its recurrence.[169] The common risk factors like hypertension, obesity, dyslipidemia,

smoking for CVD and CKD may be the cause for increase in incidence of CVD among CKD. [170,171]The spectrum of cardiovascular disease seen in CKD patients are[172]

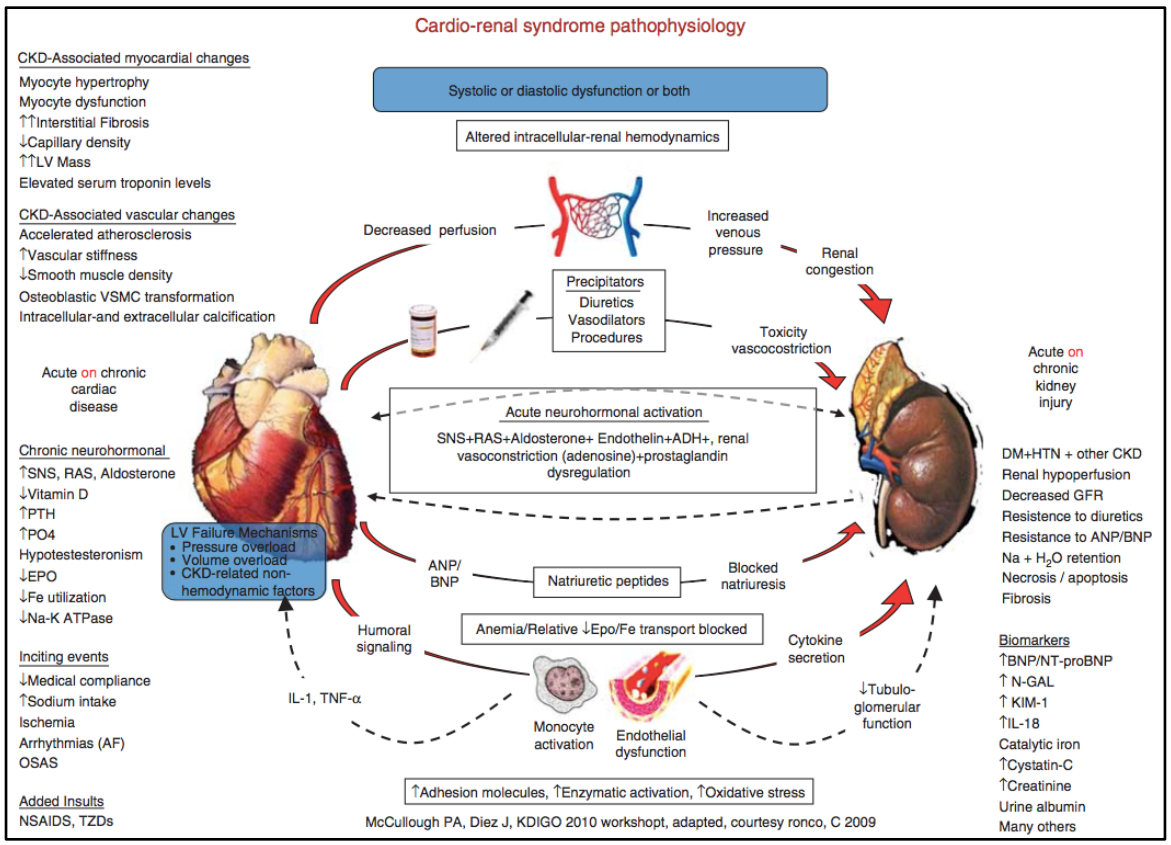
1. Coronary artery disease (CAD) and myocardial infarction (MI).
2. Congestive heart failure (CHF).
3. Peripheral arterial disease (PAD).
4. Arrhythmias.
5. Cerebrovascular disease (CVA).
6. Sudden cardiac death.

Hypertension (HT) is one of the most common cause for CVD in CKD. Activation of RAAS and sodium retention is the mechanism for developing HT in CKD. Increased sympathetic activity and elevated catecholamine concentration is seen in CKD.[173] Renalase, a soluble monooxygenase which degrades catecholamines, is expressed in renal glomeruli, PCT and cardiomyocytes is absent in CKD, so increase in catecholamine activity is seen in CKD.[174]Endothelial dysfunction is common in glomerular microalbuminuria and causes hypertension. Hypertension causes left ventricular hypertrophy causing ventricular dysfunction.[175] Asymmetric di methyl arginine (ADMA), which is a competitive inhibitor of nitric oxide synthase is increased in CKD. So there will be decreased bio availability of NO in CKD, which causes endothelial dysfunction.[176]

In CKD there will be pressure overload and volume overload that causes cardiomyopathy and left ventricular failure. Arterial stiffening due to medial and soft tissue calcification also contribute to CVD in CKD.[177] All these contribute to the cardio renal syndrome.

Figure 10,[178] gives an outline of Cardio renal syndrome

Figure 10:[178]**Pathophysiology of Cardio-renal Syndrome**



ADH-Anti Diuretic Hormone, **ANP**- Atrial natriuretic peptide, **BNP-B** type natriuretic peptide, **EPO**-Erythropoietin, **HTN** –Hypertension, **DM**-Diabetes, **LV**-left ventricular, **N-GAL**-Neutrophil gelatinase associated lipocalin, **KIM -1**- Kidney Injury molecule, **NSAID**- Non steroidal anti inflammatory drug, **OSAS**-Obstructive sleep apnoea syndrome, **PTH**-Parathyroid hormone, **SNS**-Sympathetic nervous system, **TNF-α**- Tumor necrosis factor-α, **TZD**-Thiazolidinediones, **VSMC**-Vascular smooth muscle cell.

OCCUPATION AND CKD

Several studies in different parts of the world has shown association between lower socioeconomic status (LES) and CKD. Lower socioeconomic status is defined

by educational status and occupational levels.[179,180]Low occupational level people are exposed to lot of nephrotoxins like metals (lead, mercury, silica, copper, silver etc), organic solvent, grain dust, and welding fumes (glycol ether).[181,182,183] Agricultural labourers are prone for heat stress and hence CKD. Strenuous work done in hot environment leads to volume depletion and rhabdomyolysis.[184] Our body tries to maintain the temperature at approximately 37^{o}C. Any increase in temperature stimulate the thermoregulatory center in the hypothalamus and causes vasodilatation and sweat glands stimulation and there will be regulation of temperature by convection, conduction and evaporation. There will be loss of electrolytes and extracellular fluid causing a stress to the kidneys.[185]When there is heat stress there will be release of cytokines and heat shock proteins which causes inflammatory response.[25] Persons exposed to heat stress are prone to develop both acute kidney disease(AKD) and chronic kidney disease(CKD).[185,186]In a study in El Salvador, about the Mesoamerican nephropathy (MeN), the renal biopsy showed tubulointerstitial injury and glomerulosclerosis with minimal vascular changes.[187]

Agrochemical exposure (commonly used 2,4-Dichlorophenoxyacetic acid(2,4-D), paraquat dichlorate, glyphosate) is also associated with renal dysfunction.[25] In a study among sugar cane workers for MeN have mentioned that the workers eat sugarcane and they are exposed to agrochemicals,[4] also the high fructose intake causes metabolically induced inflammatory renal damage.[4,188,189]Fructose are transported by the glucose transporters GLUT5 /GLUT2, mainly expressed in the proximal convoluted tubule. In the liver and kidney fructose is metabolized to fructose-1-phosphate by the enzyme fructokinase [Ketohexokinase(KHK)]. ThenAldose-B acts on fructose-1-phosphate to form dihydroacetone phosphate and glyceraldehyde. During the fructokinase activity there is depletion of ATP. The

depletion of ATP activates the enzyme of purine metabolism, to yield uric acid leading to hyperuricemia, oxidants and inflammatory mediators leading to tubular injury and inflammation.[188,189,190] Also when they eat sugarcane they are at risk of getting exposed to leptospiral infection.[24] Leptospirosis produces acute kidney injury, by a tubulointerstitial process with interstitial oedema and mononuclear cell infiltration. Associated electrolyte abnormalities (sodium wasting and hypokalemia) is due to the inhibition of the NaK2Cl (NKCC2) transporter in the ascending Loop of Henle.[191] In some studies, tubular dysfunction persisted and progressed to CKD when there was associated diseases like hypertension.[192]Occupational activities involving metals like lead, cadmium etc contaminate the environment. The source of lead contamination is from gasoline and paint.[193] These metals get accumulated in proximal tubule producing chronic tubulointerstitial nephritis. Nephritis may be due to oxidative stress leading to lipid peroxidation, apoptosis and necrosis.[194]

SMOKING AND CKD

Smoking is a preventable cause for many non-communicable diseases. In a metaanalysis Xia et al has stated smoking as an independent risk factor for developing CKD.[195] In patients with diabetes and hypertension smoking contributes to the progression of CKD and CVD.[196,197,198] Renal function loss was noticed in persons with history of current smoking of more than 20 cigarettes a day.[199] In diabetic nephropathy, smoking develops micro and macro albuminuria and progression to end stage renal disease.[196,198]

Mechanism of Smoking and renal disease:

Several factors affect the toxic effect of smoking on renal dysfunction. The genetic susceptibility of the individuals decide the amount of damage. The potential mediators and mechanism of smoking causing renal damage can be classified as

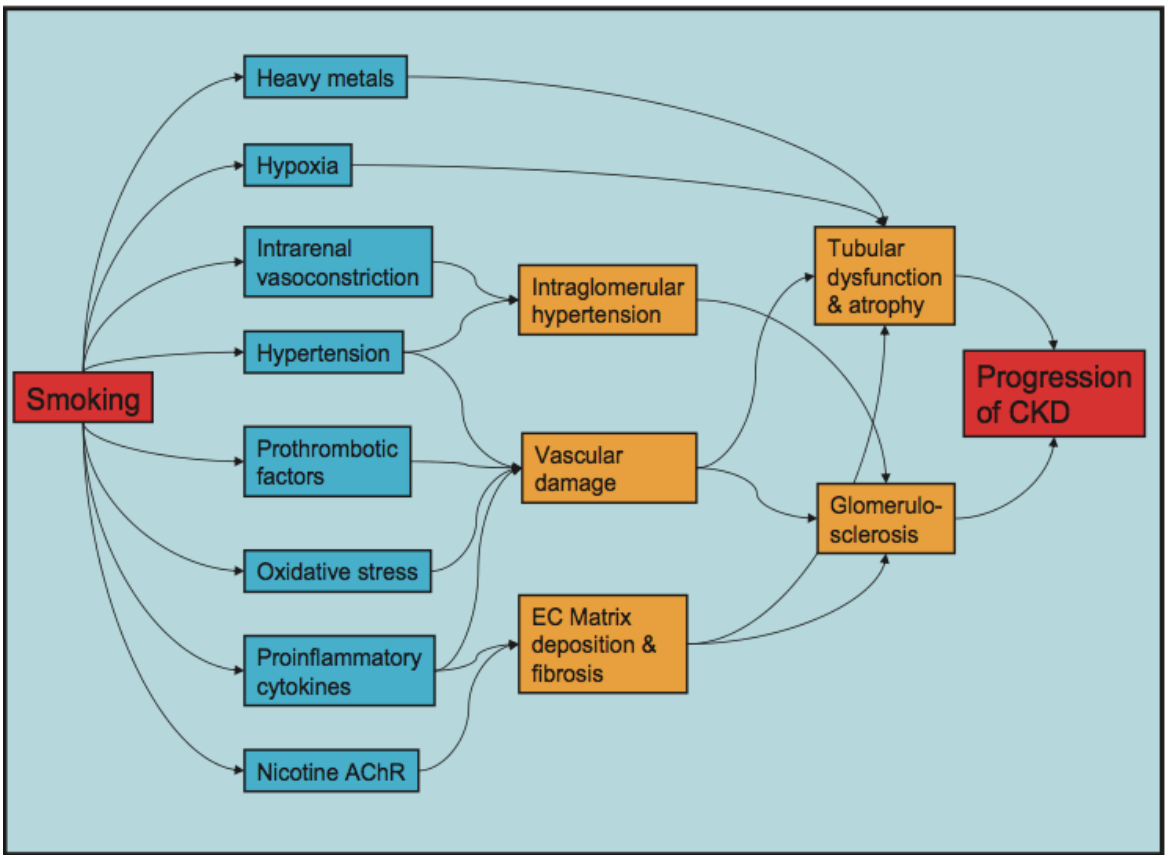
1. Non hemodynamic mechanism
2. Hemodynamic mechanism

Non Hemodynamic mechanism:

Smoking causes endothelial dysfunction, growth factor activation, oxidative stress, toxic effects on tubules, altered metabolism of lipoprotein and glycosaminoglycans and insulin resistance.[196,198] Studies have shown that nicotine induces mesangial cell proliferation and fibronectin production.[200] There is expression of nicotinic acetyl choline receptors in the mesangial cells which mediates the proliferation.[201]

Figure11,[29] explains the non hemodynamic mechanism of smoking causing renal damage.

Figure11[29] : Potential Mechanism of Smoking induced Kidney damage



AChR- Acetylcholine receptor, **EC** –Extracellular, **CKD**-Chronic Kidney Disease

Hemodynamic Mechanism:

Smoking increases the blood pressure and heart rate due to the action of nicotine present in cigarettes.[197]The increase in concentration of epinephrine and norepinephrine and direct stimulation of postganglionic nerve ending by the nicotine causes the increase in BP and heart rate.[202] In smokers there is an associated decrease in BP at night,[203]which may lead to renal dysfunction.[204] Ritz et al in their study has found that nicotine, increased the activity of sympathetic nervous system causing increased heart rate, blood pressure, intra glomerular capillary pressure leading to decrease in GFR, filtration fraction and renal plasma flow.[205] John main et al has stated that smoking causes cholesterol micro embolism causing atherosclerotic changes in renal artery and progression to end stage renal disease (ESRD).[206]

Pathological finding in kidney of smokers:

An increase in arteriolar wall thickness due to fibroelastic proliferation and intimal hyaline thickening is seen.[207] There is increased myointimal hyperplasia of intrarenal arterioles which is absent in hypertension, so smoking is the causative agent behind this change.[208] Idiopathic glomerulosclerosis is seen exclusively in smokers.[209]

ALCOHOL AND CKD

Chronic excessive alcohol intake is considered as a risk factor for liver disease, cardiovascular illness and cerebrovascular illness.[210]The association between chronic alcohol intake and kidney disease is under controversy. Experimental studies states that alcohol has an acute nephrotoxic effect.[211]Chronic alcohol consumption indirectly causes CKD by causing alcohol induced hypertension.In certain specific situations chronic alcohol dependency causes renal papillary necrosis, infection associated glomerulonephritis and acute kidney injury due to rhabdomyolysis (non

traumatic).[212,213,214]Studies report that an inverse association exist between high alcohol consumption and CKD risk.[215]The inverse association may be due to the increase in high density lipoprotein (HDLc) and plasma endogenous tissue type plasminogen activator levels and decrease in platelet aggregation by alcohol. HDLc is less atherogenic and may be responsible for the inverse association of CKD.[216,217]Animal studies state that antioxidant property of polyphenol present in red wine may reduce renal damage by the induction of glutathione peroxidase and superoxide dismutase.[218] Another animal model study has said that alcohol prevent leukocyte induced endothelial barrier damage and prevents renal ischemia.[219]

KIDNEY STONE AND CKD

Kidney stone is considered to be a risk factor for CKD. Hippisley et al have found an increased risk of ESRD among female than male kidney stone formers.[220] Some studies state that there is no relationship between renal stone and CKD.[221] Junger's et al in a study has found that 3.2% of the 1391 patients with ESRD had renal stones, and stones caused ESRD. They have found that 40% of patients with stones in the study group had solitary functioning kidney.[222] There is association of development of CKD and the kidney stone type, in a study of community stone formers struvite stones were found to be a risk factor for CKD.[223] Brushite stone type ($CaHPO_4$) is associated with glomerulosclerosis, tubular atrophy and interstitial fibrosis.[224]

Many pathways have been postulated for the development of CKD in kidney stone patients. Obstruction by stones causing obstructive uropathy can cause acute or irreversible chronic injury. There can be renal vasoconstriction and inflammation due to increase in the intratubular pressure.[225,226] In animal studies, in unilateral uretral

obstruction, osteopontin mediated interstitial influx and interstitial fibrosis have been noticed.[227]Infective struvite stones form chronic pyelonephritis, which can cause destruction of renal parenchyma.[228] In uric acid stones, the hyperuricemia causes uric acid crystals to be deposited in the interstitial tissue causing interstitial fibrosis.[229]

MATERIALS AND METHODS

STUDY CENTRE:

The study was conducted among the adult population who attended the out-patient departments at Trichy SRM Medical College Hospital and Research Centre, Irungalur, Trichy.

STUDY DESIGN:

Cross sectional Observational study

STUDY PERIOD:

One year, June 2017 to May 2018

GEOGRAPHICAL DISTRIBUTION:

Both rural and urban areas in and around Trichy.

SAMPLE SIZE:

The calculated sample size using prevalence of CKD as 17.2%, power of the study of 80% and significance level as 5% from the SEEK study[2] was 463.Atotal of 500 subjects who attended the medicine and allied out-patient department were included as study participants.

ETHICAL CONSIDERATION:

Ethical clearance for the study was obtained from the Institutional Ethical Committee(IEC) of Trichy SRM Medical College Hospital and Research Centre, Irungalur. All study subjects were explained about the study and informed written consent was obtained and the confidentiality of their results were maintained.

INCLUSION CRITERIA:

All adults of age 20 years and above, of both sex were included in the study.

EXCLUSION CRITERIA:

Patients with Acute Kidney Injury (AKI) were excluded on the basis of oliguria and rapid increase in creatinine from previous recent records.

STUDY PROTOCOL:

After enrollment in the study a detailed history was taken, which included history of presenting illness, past illness, occupation, any exposure to agrochemicals, personal history likesubstance abuse, smoking, alcohol intake was noted. The patient was enquired about history of any renal stones, cardiovascular illness, thyroid illness, diabetes mellitusand hypertension. A detailed history of treatment and drugs takenwere documented

CLINICAL EXAMINATION:

Routine clinical examination of various systems was carried out.

Blood Pressure (BP):

Blood pressure was recorded in the sitting position in the right arm using mercury sphygmomanometer. Participants whose BP ≥ 140/90 mm Hg was considered as hypertensive, JNC-7 guidelines.[230] Participants who were already on anti hypertensives were considered as hypertensive irrespective of the BP measurement.

Anthropometric measurements:

Height and weight of the study subjects were measured. Body mass index was calculated from height and weight. Height was measured using a stadiometer in centimeters. The individual was asked to stand in the upright posture barefoot, heels kept together in close approximation, with the subject's back against the vertical backboard and eyes directed forward. Weight was measured in kilograms using an electronic weighing machine, kept on a firm horizontal surface with light clothing.

Waist circumference(WC) was measured in centimeters using a non-stretchable measuring tape. WC was measured at the smallest horizontal girth between the costal margin and the iliac crest at the end of expiration. Body mass index (BMI)was calculated using the formula

BMI = Weight / height^2 (kg/m^2)

BMI Categorization WHO Guidelines :[231]

Under weight: < 18.5 kg/ m^2

Normal : 18.5 to 24.9 kg/m^2

Over weight : 25- 29.9 kg/m^2

Obese : > 30 kg/m^2

Systemic Examination was done.

Biochemical Investigation (Estimated Parameters):

Sample Collection:

An overnight 12 hours fasting venous sample was collected in a blood clot activator (BCA) tube, for analysis of fastingblood glucose, fasting lipid profile [Total cholesterol(TC), Triglycerides(TGL), High Density Lipoprotein Cholesterol(HDLc), Low Density Lipoprotein(LDLc)],thyroid function tests [Thyroid Stimulating Hormone(TSH),Free thyroxine (FT4)], Renal Function test(serum urea, serum creatinine).

A spot urine sample was collected in a clean 100ml urine collection container for spot UPCR. The samples were processed within 30 minutes to1 hour of collection. Thyroid Hormone assay was done by Electro Chemiluminescence Immunoassay (ECLIA) using Roche, cobas e411 analyzer. All other tests were performed using autoanalyser, Mindray BS 420.

Diagnosis of DM was made based on WHO criteria of fasting blood glucose level of more than 126 mg/dl.[232]Known diabetics on treatment were considered diabetic irrespective of their glycemic status.

Thyroid Function:

Euthyroid state was diagnosed with TSH – 0.4 – 4.2μIU/L and FT4 level 0.8- 2 ng/dl. Overt hypothyroidism was diagnosed with TSH ≥ 5μIU/L and Free T4 level < 0.8 ng/dl; Subclinical hypothyroidism was diagnosed when TSH ≥ 5μIU/L and with normal Free T4 levels. Hyperthyroidism was diagnosed when TSH< 0.25μIU/L and Free T4>2 ng/dl. Subclinical Hyperthyroidism was considered when TSH< 0.25μIU/L and Free T4 within normal range. Patients who were on treatment with Thyroxine supplementation were considered as hypothyroid.

Renal Function:

Serum Urea and Serum Creatinine were measured.

Estimated Glomerular Filtration Rate (eGFR) :

Using serum creatinine Estimated Glomerular Filtration Rate (eGFR) was calculated using Chronic Kidney Diseases- Epidemiology Collaboration group (CKD-EPI) formula[49].

The CKD-EPI equation, expressed as a single equation

$$\mathbf{GFR = 141*min\ (Scr/K,1)^{\infty *}max(Scr/K,1)^{-1.209*}0.993^{Age*}1.018[if\ female]^{*}1.159[if\ black]}$$

Scr is serum creatinine(mg/dl), K is 0.7 for female and 0.9 for males,∞ is -0.329 for females and -0.411 for males, min indicates the minimum of Scr/K or 1, and max indicates the maximum of Scr/K or 1.

Staging of CKD was done as per GFRcategory based on KDIGO 2012[39]

G1 - eGFR ≥ 90ml/min/1.73 m^2 – Normal or High

G2 - eGFR 60-89ml/min/1.73 m^2 – Mildly decreased

$G3_a$ - eGFR 45-59ml/min/1.73 m^2 – Mildly to moderately decreased

$G3_b$ - eGFR 30-44ml/min/1.73 m^2 – Moderately to severely decreased

G4 - eGFR 15-29ml/min/1.73 m^2 – Severely decreased

G5- eGFR <15ml/min/1.73 m^2 - Kidney failure

CKD was defined as eGFR <60ml/min/1.73 m^2

Urine Spot Protein- Creatinine Ratio(UPCR) Categorisation[35]

1. Normal, P1 - UPCR < 0.15
2. Moderate proteinuria, P2 - UPCR > 0.15-0.5
3. Overt Proteinuria, P3 - UPCR > 0.5

Cardiovascular disease was considered present or absent based on the diagnosis made by Physician or Cardiologist.

METHODOLOGY

ESTIMATION OF PLASMA GLUCOSE

Method:

Glucose Oxidase Peroxidase method (GOD-POD) (End point)

Principle:

Plasma glucose is first oxidized to Gluconic acid by the enzyme Glucose Oxidase(GOD)with release of Hydrogen peroxide, which is further converted into nascent oxygen and water by the enzyme peroxidise (POD). 4-Aminoantipyrine takes up the oxygen, simultaneously with phenol,forms a pink coloured chromogen, measured at 505 nm.

$$\text{Glucose} + O_2 \xrightarrow{\text{GOD}} \text{Gluconic acid} + H_2O_2$$

$$2\ H_2O_2 + \text{4-Aminoantipyrine+Phenol} \xrightarrow{\text{POD}} \text{Quinoneimine+ 4 } H_2O$$

Reagents:

Glucose Oxidase	:	≥10 KU/L
Peroxidase	:	≥ 1 KU/L
Phosphate buffer (pH) 7.5	:	250mmol/L
Phenol	:	5 mmol/L
4-Aminoantipyrine	:	0.5 mmol/L
Standard concentration	:	100 mg/dl

Procedure:

Take three test tubes and label as Blank (B), Test (T) and Standard (S) as follows, Incubation period: 10 min,Temperature at 37^0C.Mix well and read absorbance of Test (T) and Standard(S) against distilled water at 505 nm

S.No	Reagent	Blank	Standard	Test
1	Glucose Reagent	1.0 ml	1.0 ml	1.0 ml
2	Glucose Standard	--	10µl	--
3	Specimen	--	--	10 µl

Calculation:

Glucose concentration (mg/dl) =(Δ Abs-Test / Δ Abs-Standard) x100

Reference value:

Plasma Fasting Glucose : 70-100 mg/dl

Post Prandial Plasma Glucose: < 140 mg/dl

ESTIMATION OF SERUM UREA

Method:

Urease –Glutamate Dehydrogenase("Urease-GLDH")

Principle:

Urea is hydrolysed into ammonia and carbon dioxide by the presence of water and urease. The ammonia combines with 2-oxoglutarate and NADH in the presence of glutamate-dehydrogenase (GLDH) to produce glutamate and NAD^+. The GLDH is the rate limiting enzyme. The rate of decrease in NADH is proportional to the concentration of urea, within the given time intervals. As the kinetic is very fast, this test is preferably designed for analyzer application.

$$\text{Urea} + 2H_2O \xrightarrow{\text{Urease}} 2NH_4^+ + 2\,HCO_3^-$$

$$\text{2-Oxoglutarate} + NH_4^+ + \text{NADH} \xrightarrow{\text{GLDH}} \text{L-Glutamate} + NAD^+ + H_2O$$

Reagents:

1. Enzymes:

Tris buffer (pH 7.8)	:	125 mmol/L
Urease	:	≥ 20 kU/L
GLDH	:	≥ 0.3 kU/L
ADP	:	0.88 mmol/L
Sodium Azide	:	0.095 %

2. Substrates:

2-oxoglutrate	:	25 mmol/L
NADH	:	1.25 mmol/L

Sodium Azide : 0.095%

3.Standard:

Urea : 80mg/dl

Sodium Azide : 0.095%

Preparation of reagent:

Reagent is prepared by mixing 4 parts of enzymes with 1 part of substrates.

Specimen:

Serum

Assay:

Wavelength : 334nm, 340nm, 365nm

Temperature : 25°C, 30°C or 37°C

Optical path : 1cm

Measurement : Against reagent blank(RB).

Only one reagent blank, per series is required.

Procedure:

Sample Start:

	Reagent blank(RB)	Sample / standard
Sample / standard	-	10µl
Working(mono) reagent	1000µl	1000µl
Mix and read absorbance of Sample /standard after 30 seconds at 37 ^{0}C (A1), and read after exactly 1 minute(A2). Calculate the absorbance difference: ΔA Sample /standard= (A2-A1)-ΔARB		

Substrate Start Procedure:

	Blank	**Standard**
Sample /standard	-	10µl
Reagent 1	1000µl	1000 µl
Mix, incubate approximately for 5 minute.		
Reagent 2	250µl	250µl
Mix, read absorbance of Sample /standard after 60 seconds and read after exactly 1 minute (A2). Calculate the absorbance difference: ΔA Sample /standard= (A2-A1)-ΔARB.		

Calculation:

C= 80.0× ΔA Sample/ ΔA standard [mg/dl]

Measuring range:

The test has a measuring range of 2 to 300mg/dl or 0.3to 50mmol/l.

Reference values:

Serum: 10-50 mg/dl

ESTIMATION OF SERUM CREATININE

Method:

Modified Jaffe's kinetic method

Principle:

Creatinine, in the sample reacts with picric acid in alkaline medium to form creatinine picrate (red coloured complex), measured at 500 nm.

Creatinine + Picricacid ⟶ Creatinine picrate complex

Specimen:

Serum, Urine

Reagents:

Standard	:	2 mg/dl

Reagent-1

Sodium Hydroxide	:	0.2 mol / L

Reagent 2

Picric acid	:	20 mmol / L

Storage: 2º-8ºC

Preparation of working solution:

The reagents are brought to room temperature. 4 parts of reagent 1 and 1 part of reagent 2 are mixed.

General system parameters:

Reaction slope	:	Increasing
Reaction Type	:	Fixed Time
Flow cell Temp.	:	25°C, 30°C or 37°C
Wavelength	:	500 nm (490-510 nm)
No. of Readings	:	120 sec.

Delay Time	:	30 sec.
Reagent Volume	:	1.0 ml
Sample Volume	:	100 µl
Std. Concentration	:	2 mg/dl
Path length	:	1 cm
Zero Setting With	:	Distilled water

The instrument is set with above system general parameters.

Procedure:

The sample and working solution are allowed to attain room temperature, prior to use Three test tubes are taken and labelled as Standard (S), Blank (B), Test (T). 1 ml of working reagent is added to all three test tubes. 100µl of sample is added to test tube 'T' and 100µl of standard is added to test tube 'S'. Then mixed and reading is taken absorbance A1 after 60 seconds and A2 after further 120 seconds. ΔA = A2-A1. For urine spot creatinine dilute urine 1 +49 with distilled water, multiply the result by 50.

	Blank	**Standard**	**Test**
Reagent	1ml	1ml	1ml
Standard	-	100µl	-
Sample	-	-	100µl

Calculation:

Serum creatinine(mg/dl) =Concentration of standard× (ΔA Sample/ ΔA standard)

Urine spot creatinine(mg/dl) = Concentration of standard× (ΔA Sample/ ΔA standard)

Linearity:

This method is linear up to 10 mg/dl

Reference range:

Serum Creatinine:

Males : 0.9-1.3 mg/dl

Females : 0.6-1.1mg/dl

Spot Urine Creatinine, morning sample

Male : 39-259 mg/dl

Female: 28-217 mg/dl

LIPID PROFILE

ESTIMATION OF SERUM TOTAL CHOLESTEROL (TC)

Method

"CHOD-POD"–Cholesterol Oxidase Peroxidase.

Principle:

Determination of cholesterol after enzymatic hydrolysis and oxidation. Then photometric estimation of cholesterol is done by coupling reactions with free cholesterol. The calorimetric indicator is quinoneimine(red coloured complex), generated from 4-aminoantipyrine and phenol by hydrogen peroxide under catalytic action of peroxidase (Trinder's reaction)

The reactions are as follows:

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{Chol.Esterase}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + \tfrac{1}{2} O_2 + H_2O \xrightarrow{\text{Chol.Oxidase}} \text{Cholest-4en-3-one} + H_2O_2$$

$$2\ H_2O_2 + \text{4- Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + 4\ H_2O$$

The intensity of the red colour is proportional to the concentration of cholesterol in the serum.

Reagents

Good's buffer (pH 6.7)	:	50mmol/L
Phenol	:	5 mmol/L
4-Aminoantipyrine	:	0.3 mmol/L
Cholesterol esterase	:	≥ 200 U/L
Cholesterol oxidase	:	≥50 U/L

Peroxidase	:	≥3 kU/L
Standard	:	200 mg/dl (5.2 mmol/L)

Reagent and standard are stable if stored at 2- 8^0C

Specimen

Serum

Preparation of working solution:

Ready to use reagents and standard are allowed to attain room temperature.

Procedure:

Prior to use, the sample and working solution are brought to room temperature.

System parameters:

Reaction Type	:	End point assay
Reaction slope	:	Increasing
Flow cell Temp.	:	Room temperature / 37°C
Sample Volume	:	10 µL
Reagent Volume	:	1 mL
Delay Time	:	5 sec
Wavelength	:	500 ± 10 nm
Optical Path	:	10 mm
Standard Concentration	:	200 mg/dl
Zero Setting	:	With Distilled water

Procedure:

Three test tubes are labelled as blank (B),Test (T) and Standard (S), one ml of working reagent is added to all 3 test tubes. 10µL of standard is added to test tube 'S'and 10µL of sample is added to test tube 'T'. Then it is mixed, incubated for 10 min at room temperature.

	Blank	Standard	Test
Distilled water	10µl	--	--
Reagent	1ml	1ml	1ml
Standard	--	10µl	--
Sample	--	--	10µl

Calculation:

$$\text{Cholesterol (mg/dl)} = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times \text{Concentration of standard}$$

Measuring range:

Linearity is up to 3-750mg/dl.

Lower limit of detection: 3 mg/dl

Reference values

Serum Total Cholesterol:

Desirable : < 200 mg/dl

Borderline : 200 - 240 mg/dl

High : > 240 mg/dl

ESTIMATION OF SERUM TRIGLYCERIDES(TGL)

Method:

Colorimetric Enzymatic test using Glycerol-3- phosphate oxidase (GPO)

Principle

Estimation is based on enzymatic splitting with lipoprotein lipase. The glycerol formed is converted to Glycerol – 3- phosphate which is further oxidised to Dihydroacetone phosphate and Hydrogen peroxide. The oxygen formed by peroxidase is accepted by 4-aminoantipyrine in the presence of 4-chlorophenol to form Quinoneimine (red colour complex) which is measured at 500nm.

$$\text{Triglycerides} \xrightarrow{\text{Lipoprotein Lipase}} \text{Glycerol + Fatty acid}$$

$$\text{Glycerol + ATP} \xrightarrow{\text{Glycerol Kinase}} \text{Glycerol -3-phosphate + ADP}$$

$$\text{Glycerol -3-phosphate} + O_2 \xrightarrow{\text{Glycerol -3-Phosphate Oxidase}} \text{Dihydroxyacetone phosphate} + H_2O_2$$

$$2H_2O_2 + \text{Aminoantipyrine + 4- Chlorophenol} \xrightarrow{\text{Peroxidase}} \text{Quinoneimine} + HCl + 4\ H_2O$$

Reagent:

4-Chlorophenol	:	4 mmol / l
Mg^{2+}	:	15mmol/l
ATP	:	2 mmol / l
4-Aminoantipyrine	:	0.5mmol/l

Glycerolkinase	:	≥0.4 kU/l
Peroxidase	:	≥ 2 kU/l
Lipoprotein lipase	:	≥ 2 kU/l
Good's buffer pH7.2	:	50 mmol / l
Glycerol-3-phosphate–oxidase:		≥0.5 kU /l
Standard	:	200mg/dl (2.3 mmol/l)

Specimen:

Serum

Storage: at 2ºC-8ºC

General system parameters:

Reaction Type	:	End point
Wavelength	:	500 nm
Reaction slope	:	Increasing
Delay Time	:	5 seconds.
Flow cell Temp.	:	Room temperature or 37°C
Reagent Volume	:	1.0 ml
Sample Volume	:	10 µl
Std. Concentration	:	200 mg/dl
Optical Path	:	1 cm

Zero Setting with Distilled water

The instrument is set with above system parameters.

Procedure

The sample and working solution are brought to room temperature. Three test tubes are taken and labelled as Standard (S), Test (T), blank (B). One ml of working reagent is added to three test tubes. 10µL of sample is added to test tube labelled as 'T' and 10µL of standard is added to test tube labelled as 'S'. Then mixed and incubated 10 minutes at room temperature.

	Blank	**Standard**	**Test**
Distilled water	10µl	--	--
Reagent	1ml	1ml	1ml
Standard	--	10µl	--
Sample	--	--	10µl

Calculations:

$$\text{Triglyceride (mg/dl)} = \frac{\text{Absorbance of Sample} \times \text{Concentration of standard}}{\text{Absorbance of Standard}}$$

To correct for free glycerol subtract 10mg/dl from the triglycerides value.

Measuring range:

This method is used from the measuring range of 2-1000mg/dl.

Lower limit of detection is 2mg/dl

Reference values:

Serum Triglycerides:

Normal : < 200 mg/dl

Borderline High : 200-400mg/dl

Elevated : >400 mg/dl

ESTIMATION OF SERUM HIGH DENSITY LIPOPROTEI CHOLESTEROL (HDLc)

Method

Direct enzymatic method

Principle:

Antibodies against human lipoproteins are utilized to form antigen-antibody complexes with chylomicrons, LDL, VLDL and in a way that only HDL-cholesterol is selectively estimated by an enzymatic cholesterol measurement.

The reaction sequence is, as follows:

$$\text{LDL, VLDL, Chylomicrons} \xrightarrow{\text{Anti-human } \beta \text{ – lipoprotein antibodies}} \text{Antigen – antibody complexes +HDL}$$

$$\text{HDL –Cholesterol} + H_2O + O_2 \xrightarrow{\text{CHE \&CHO}} \text{Cholest-4-en-3-one + Fatty acid} + H_2O_2$$

$$H_2O_2 + \text{F- DAOS} + \text{4- Aminoantipyrine} \xrightarrow{\text{Peroxidase}} \text{Blue Complex} + H_2O$$

CHE – Cholesterol esterase; CHO-Cholesterol oxidase; F-DAOS-N-Ethyl-N-(2-Hydroxy-3-sulfopropyl)-3,5-Dimethoxy-4-Fluoroaniline, sodium salt

Reagents:

Reagent 1:

Good's buffer pH 7.0 : 25 mmol / L

Peroxidase : 2000 U/L

Ascorbate oxidase : 2250 U/ L

Anti-human β – lipoprotein Antibody (sheep)

4-Aminoantipyrine : 0.75mmol/L

Reagent 2:

Good's buffer pH 7.0	:	30 mmol / L
Cholesterol oxidase	:	20000U/L
Cholesterol esterase	:	4000 U/L

N-Ethyl-N-(2-hydroxy-3-sulfopropyl): 0.8mmol/L

Sodium salt (F-DAOS), 3, 5- dimethoxy-4-fluoroaniline.

Calibrator:

HDL-Cholesterol 50.6 mg/dl

Storage: at 2º-8ºC

Specimen:

Serum

General system parameters:

Reaction Type	:	End point
Wavelength	:	600/700 nm
Reaction slope	:	Increasing
Delay Time	:	5 seconds
Flow cell Temp.	:	Room temperature or 37°C
Reagent 1 Volume	:	240 µL
Reagent 2 Volume	:	60 µL
Sample Volume	:	2.4 µL
Calibrator	:	50.6 mg/dL
Zero Setting	:	with Distilled water
Optical Path	:	1 cm

Procedure:

The sample and working solution are allowed to attain room temperature prior to use Three test tubes are labelled as Test (T), Calibrator (C), blank (B), 240 µL of working reagent 1 is added to all three test tubes. 2.4µL of sample is added to Test 'T' and 2.4 µL of calibrator is added to Calibrator 'C'. Then mixed and incubated for 5 minutes at room temperature. Read the absorbance A_1, then 60 µL of working reagent 2 is added to all three test tubes. It is mixed and incubated for 5 minutes at room temperature. Read the absorbance $A_{2.}$ $\Delta A = (A_2 - A_1)$ sample or calibrator.

	Blank	**Calibrator**	**Test**
Calibrator	--	2.4 µL	--
Sample	--	--	2.4 µL
Distilled water	2.4 µL	--	--
Reagent 1	240 µL	240µL	240 µL
Mixed, incubated 5min at 37°C,read absorbance A_1Then add:			
Reagent 2	60 µL	60µL	60µL
Mixed, incubated 5 min at 37°C, read absorbance $A_{2.}$			

Calculation:

$$\text{HDLc (mg/dl)} = \frac{\Delta A \text{ Sample}}{\Delta A \text{ Calibrator}} \times \text{Calibrator concentration}$$

Measuring range:

This method is used to determine HDL-c concentration within a measuring range from 1-180mg/dl.

Lower limit of detection is 1 mg/dl

Reference values:

Serum HDL-C

Low : < 40 mg/dl

Desirable : 40-60 mg/d

ESTIMATION OF SERUMLOW DENSITY LIPOPROTEIN CHOLESTEROL

Method:

Direct enzymatic method

Principle

In the first step, LDL is selectively protected while non-LDL-lipoprotein are enzymatically processed. In the second step, LDL is released and LDL-cholesterol is selectively determined in a blue colour producing enzymatic reaction.

The reaction sequence is as follows:

1. LDL + Reagent 1 $\longrightarrow$ Protected LDL

$$\text{HDL, VLDL, Chylomicrons} \xrightarrow{\text{CHE \&CHO}} \text{Cholestenone} + H_2O_2$$

$$H_2O_2 \xrightarrow{\text{Catalase}} H_2O$$

2. Protected LDL + Reagent 2 $\longrightarrow$ LDL

$$\text{LDL-C} \xrightarrow{\text{CHE \&CHO}} \text{Cholestenone} + H_2O_2$$

$$H_2O_2 + \text{4-Aminoantipyrine} + \text{H-DAOS} \xrightarrow{\text{POD}} \text{Blue Colour}$$

CHE – Cholesterol esterase ; CHO-Cholesterol oxidase

Reagents:

Reagent 1:

Cholesterol oxidase (CHO)	:	≥2.5 kU/L
Cholesterol esterase (CHE)	:	≥2.5 kU/L
Catalase	:	≥500 kU/l
N-(2-hydroxy-3-sulfopropyl)	:	0.5mmol/L
3, 5- dimethoxyaniline, (H-DAOS)		
Good's buffer pH 6.8	:	20 mmol / L

Reagent 2:

4-Aminoantipyrine	:	3.4mmol/L
Peroxidase (POD)	:	≥15 kU/L
Good's buffer pH 7.0	:	25 mmol / L

Calibrator:

LDL-Cholesterol 132 mg/dL

Storage: at 2ºC-8ºC

Preparation of working solution

The reagents are brought to room temperature. Both reagent and standard are supplied as ready to use.

Sample

Serum

General system parameters:

Reaction Type	:	End point
Wavelength	:	600/700 nm (bichromatic measurement)
Reaction slope	:	Increasing
Delay Time	:	30 seconds.

Flow cell Temp. : 37°C

Sample Volume : 3.0 µL

Reading Time : 180 seconds.

Optical Path : 1 cm

Reagent 1 Volume : 280 µL

Reagent 2 Volume : 70 µL

Calibrator : 132 mg/dL

Zero Setting with Distilled water

The instrument is set with above system parameters.

Procedure:

Three test tubes are labelled as Test (T), Calibrator (C), blank (B). 280 µL of working reagent 1 is added to all three test tubes. 3.0µL of sample is added to 'T' and 3.0 µL of calibrator is added to 'C'. Then mixed and incubated for 5 minutes at room temperature. Read the absorbance A_1, then 70 µL of working reagent 2 is added to three test tubes. It is mixed well and incubated for 5 minutes at room temperature. Read the absorbance A_2.

$\Delta A = (A_2 - A_1)$ sample or calibrator- $(A_2 - A_1)$ blank.

	Blank	**Calibrator**	**Test**
Calibrator	--	3.0 µl	--
Sample	--	--	3.0 µl
Reagent 1	280 µl	280µl	280 µl
Distilled water	3.0 µl	--	--
Mix, incubate 5min at 37°C,read absorbance A_1Then add:			
Reagent 2	70 µl	70µl	70µl
Mix, incubate 5 min at 37°C, read absorbance A_2.			

Calculation:

$$\text{LDLc (mg/dl)} = \frac{\Delta A \text{ Sample}}{\Delta A \text{ Calibrator}} \times \text{Calibrator concentration}$$

Measuring range:

This method is used to determine LDL-C concentration within a range from 1-400mg/dl.

Reference values:

Serum LDL-C:

Desirable	: ≤ 130 mg/dL
Borderline	: 130 - 160 mg/dL
High	: > 160 mg/Dl

SPOT URINE PROTEIN ESTIMATION

Method:

Photometric test using pyrogallol red.

Principle:

Proteins form a red complex with pyrogallol red /molybdate. The absorbance is directly proportional to the protein concentration.

Reagents:

Pyrogallol red	:	60μmol/L
Sodium molybdate	:	40μmol/L
Standard	:	1300 mg/L

Storage: 2-8^{o}C, do not freeze the reagent

Specimen:

Spot Urine

Stability:

At 20-25^{o} C	:	1 day
At 4-8^{o}C	:	7 days
At -20^{o}C	:	1 month

Assay procedure:

Wavelength	:	600 nm
Optical path	:	1 cm
Temperature	:	37^{o}C
Measurement	:	Against reagent blank

Procedure :

Take three test tubes label them as Blank (B), Test (T), Standard (S).Take 1000µl of reagent to the three test tubes .Add 20µl of distilled water to the blank(B), 20µl of sample to the test(T) and 20µl of standard to the standard (S)

	Blank	**Standard**	**Test**
Reagent	1000µl	1000µl	1000µl
Distilled water	20µl	-	-
Standard	-	20µl	-
Sample	-	-	20µl

Mix and incubate for 10 minutes and read absorbance against reagent blank

Calculation

With standard or calibrator

Spot urine protein (mg/L) = (A Sample /AStandard) x Conc. Standard (mg/L)

Measuring range:

Range 20-3000 mg/L

The lower limit of detection 20 mg/L

ESTIMATION OF THYROID STIMULATING HORMONE (TSH)

Method:

Electro Chemiluminescence Immunoassay (ECLIA)

Assay Principle:

Sandwich principle

- First incubation: 50μL of sample, a biotinylated monoclonal TSH specific antibody and a monoclonal TSH specific antibody labelled with a ruthenium complex to form a sandwich complex.
- Second incubation: Streptavidin coated microparticles bind to the complex by interaction between biotin and streptavidin
- The reaction mixture is aspirated into the measuring cell where the microparticles are magnetically captured on to the surface of the electrode.Unbound substances are removed with Procell. Application of a voltage to electrode then induces chemiluminescent emission which is measured by a photomultiplier
- Results are determined via a calibration curve which is instrument specifically generated by two point calibration and master curve provided via a reagent barcode

Reagent- working solution:

The reagent pack is labelled as TSH

M Streptavidin coated micro particles transparent cap 1 bottle,12 ml

Streptavidin coated micro particles 0.72mg/ml, preservative

R1 Anti –TSH- Ab-biotin(gray cap) 14 ml; Biotinylated monoclonal anti-TSH antibody (mouse) 2 mg/L; phosphate buffer 100mmol/L, pH 7.2; preservative

R2 Anti –TSH- Ab-Ru(bpy) (black cap) 12 ml; Monoclonal anti-TSH antibody (mouse/human). Labeled with ruthenium complex 1.2 mg/L; phosphate buffer 100mmol/L. pH 7.2; preservative

Storage and stability: 2-8 ^{0}C, store upright, so that the micro particles are completely available during automatic mixing. Do not freeze.

Specimen:

Serum

Calibration:

This method has been standardized against the 2ndIRP WHO reference standard 80/558

Calibration is performed once per reagent lot using fresh reagent.

Measuring range:

Measuring range is 0.005-100 μIU/ml

Lower detection limit 0.005 μIU/ml

Values below lower detection limit is reported as < 0.005 μIU/ml and above the measuring range is given as > 100 μIU/ml.

Reference range : 0.4 - 4.2μIU/ml

ESTIMATION OF FREE THYROXINE (FT4)

Method:

Electro Chemiluminescence Immunoassay (ECLIA)

Assay Principle:

Competition principle

A specific anti-T4 antibody labeled with a ruthenium complex [Tris (2,2[1]-bipyridyl)ruthenium(II)-complex (Ru(bpy))] is used to determine free thyroxine is used.

- First incubation: 15 μL of sample and a T4 –specific antibody labeled with a ruthenium complex.
- Second incubation: After addition of the biotinylated T4 and streptavidin –coated micro particles, the still-free binding sites of labelled antibody become occupied with formation of an antibody-hapten complex. The entire complex interact via biotin and streptavidin.
- The reaction mixture is aspirated into the measuring cell where the microparticles are magnetically captured onto the surface of electrode. Unbound substances are then removed with Procell/Procell M.Application of voltage then induces chemiluminescent emission which is measured by auto multiplier.
- Results are determined via a calibration curve which is instrument specifically generated by 2 point calibration and a master curve provided via the reagent bar code.

Reagent-working solutions:

M- Streptavidin –coated micro particles 12ml;Streptavidin-coated micro particles 0.72 mg/ml;Preservative.

R1-Anti-T4-Ab~Ru(bby)- 18 ml: polyclonal anti-T4-antibody (sheep) labelled with ruthenium Complex 75 ng/ml; phosphate buffer 100mmol/L, pH7.0; Preservative.

R2- T4~biotin 18ml; biotinylated T4 2.5ng/ml ;phosphate buffer 100mmol/L, pH 7.0; Preservative

Storage and stability: 2-8 ^{0}C, Store upright, so that the microparticles are completely available during automatic mixing. Do not freeze.

Specimen:

Serum

Calibration:

This method is standardized against the the Elecsys FT4 method.Calibration must be performed once per fresh reagent lot.

Calculation: The analyzer calculates the analyte concentration of each sample either in pmol/L, ng/dl or ng/L.

Measuring range:

Measuring range = 0.3-100pmol/L.

Limit of Blank=0.3pmol/L

Limit of detection=0.5pmol/L

Limit of Quantitation= 3pmol/L with a total allowable error of ≤30%

Values below limit of detection are reported as <0.3pmol/L.

Values above the range are reported as >100pmol/L

Conversion formula pmol/Lx0.077688=ng/dl

Reference range: 0.8-2 ng/dl

STATISTICAL ANALYSIS

Data was entered in Microsoft Office Excel and data was analyzed by using Statistical Package for Social Sciences(SPSS) software, version 21.0.

Prevalence of CKD and other risk factors were expressed as percentage. Continuous variables like Body Mass Index(BMI), Blood Pressure (B.P), Blood glucose, estimated Glomerular Filtration Rate(eGFR), Serum Lipid Profile, free Triiodothyronine(FT3), free Thyroxine(FT4) which had normal distribution were presented as mean along with the standard deviation (SD). For continuous variables which had non normal distribution like Thyroid stimulating hormone(TSH), Serum Urea, Serum Creatinine and Urinary Protein Creatinine Ratio(UPCR) median and Inter Quartile Range(IQR) were used. Categorical variables such as gender were presented as percentage. Student ‘t’ test was used to analyze the difference in the mean between two groups. Chi square test was used to analyze the presence of association between the outcome variable (Stage1-2 / Stage 3a-5, CKD) and other independent variables.

RESULTS

A total of 500 individuals were included in the study. Of the study population there were 211(42.2%) females and 289(57.8%) males. The mean age of the study population was 48.31±12.76 years. There were 83(16.6%) participants who were farmers by occupation. Among the 500 study population 22(4.4%) gave a history of exposure to agrochemicals, 11(2.2%) had renal calculi, 68(13.6%) gave a history of smoking and 104(20.8%) were alcoholics.

The mean eGFR of the study population was 74.3± 23.88 ml/min/1.73 m^2.The study population was categorised based on their CKD stages, as CKD stage (1 and 2) whose eGFR was above 60 ml/min/1.73 m^2 and CKD (stages 3a, 3b, 4 and 5), whose eGFR was less than 60 ml/min/1.73 m^2.

Further results are presented under the following headings,

A) Age and gender wise distribution of eGFR among study population.
B) Prevalence of various UPCR stages among the study population and their association with different stages of CKD.
C) Prevalence of CKD stages based on eGFR.
D) Association of thyroid function and lipid profile with different stages of CKD.
E) Association of risk factors like BMI, DM, HT and CVD with different stages of CKD.

A) Age and gender wise distribution of eGFR among study population

Table 3: Age and gender wise distribution of mean eGFR

Age	Male n (%)	Female n(%)	Number of patients n(%)	eGFR (Mean±SD) ml/min/1.73m^2
20-29 years	21(48.8)	22(51.2)	43(8.6)	94.76±21.01
30-39 years	46(57.5)	34(42.5)	80(16)	87.40±19.13
40-49 years	67(53.2)	59(46.8)	126(25.2)	78.78±21.88
50-59 years	89(61.8)	55(38.2)	144(28.8)	68.24±22.45
60-69years	49(60.5)	32(39.5)	81(16.2)	60.88±20.42
>70 years	17(65.4)	9(34.6)	26(5.2)	53.73±16.25
Total	289(57.8)	211(42.2)	500(100)	74.30±23.88

Table 3, shows the distribution of mean eGFR of the study group by age and gender.

Among the study population there were 211 (42.2%) females and 289 (57.8%) males. The number of males were comparatively more than females in each age group, except for age group (20-29 years) female participants were more than males. The average eGFR among the age group of 20-29 years(n=43 patients) was 94.76 ± 21.01,which was normal. There was decreasing trend in eGFR as age increases. Among the individuals aged >70 years, the eGFR was 53.73±16.25 ml/min/1.73m^2 which was lowest among the study population. The mean eGFR of the males was 73.03±24.76 ml/min/1.73 m^2. The mean eGFR of females was 76.03±22.54 ml/min/1.73 m^2.

Table 4: Baseline Characteristics of the study population

Variables	CKD stage 1-2 Mean±SD/Median(IQR)	CKD stage 3a-5 Mean±SD/Median(IQR)	pvalue
Age(in years)	45.6±12.1	57.6±10.46	0.038*
BMI(kg/ m^2)	25.69±4.88	25.71±4.87	0.96
WC(cm)	93.23±11.19	93.99±12.72	0.02*
SBP(mmof Hg)	120.88±16.87	129.29±21.58	0.001*
DBP(mm of Hg)	78.24±10.32	80.21±12.32	0.088
FBS(mg/dl)	123.24±65.88	127.67±60.31	0.589
TGL(mg/dl)	125.75±71.30	149.17±79.23	0.003*
TC(mg/dl)	168.4±47.36	159.17±45.00	0.066
LDLc(mg/dl)	101.23±30.86	94.70±29.64	0.046*
HDLc(mg/dl)	39.71±9.48	37±11.01	0.010*
FT4(ng/dl)	1.28±0.50	1.31±0.74	0.046*
TSH(µIU/L)	2.54(1.9)	2.62(2.49)	0.268
Urea(mg/dl)	21.00(6)	33.00(20)	0.000*
Creatinine(mg/dl)	0.98(0.3)	1.5(0.84)	0.000*
UPCR	0.2(1.1)	0.12(0.14)	0.000*

*pvalue < 0.05 is statistically significant

BMI- Body Mass Index, **SBP**- Systolic Blood Pressure, **DBP**-Diastolic Blood Pressure, **FBS**- Fasting Blood Sugar, **TGL**- Triglycerides, **TC**- Total Cholesterol, **LDLc**- Low Density Lipoprotein Cholesterol, **HDLc**- High Density Lipoprotein Cholesterol, **TSH**- Thyroid Stimulating Hormone, **FT4**- Free Thyroxine, **FT3**- Free Triiodothyronine, **UPCR**- Urinary Protein Creatinine Ratio

Table 4, shows the baseline characteristics ofthe study population. Mean values of age, WC, SBP, TGL, LDLc, HDLc, FT4, Sr.Urea, Sr.Creatinine and UPCR, significantly differed between the different stages of CKD. The mean values of other parameters BMI, DBP, FBS, TC, TSH did not show any statistical significance between the different stages of CKD.

B) Prevalence of various UPCR stages among the study population and their association with different stages of CKD.

B- 1) Table 5: Prevalence of UPCR stages among study population

Spot Urine PCR	Number of patient(n)	Percentage (%)
P1 (< 0.15)	302	60.4
P2 (0.15-0.5)	133	26.6
P3 (> 0.5)	65	13
Total	500	100

As seen in table 5, among the 500 study population 302 (60.4%) had a spot urine PCR of < 0.15 which was normal(P1), 133 (26.6%) study participants had a UPCR between 0.15 and 0.5 which was moderate proteinuria(P2), and 65 (13%) had UPCR more than 0.5 which was overt proteinuria(P3).

B-2) Table6: Association of UPCR with different stages of CKD

UPCR Category	CKD stage 1- 2 n(%)	CKD stage3a-5 n(%)	Chi-square value	P value
P1(< 0.15)	260(86.1)	42(13.9)	61.20	**0.000***
P2(0.15-0.5)	100(75.2)	33(24.8)		
P3(>0.5)	27(41.5)	38(58.5)		
Total	387(77.4)	113(22.6)		

*pvalue < 0.05 is statistically significant

Table 6, shows the association between spot UPCR and different stages of CKD. In the CKD (stage1-2) 86.1% had normal protein excretion, 75.2% had moderate proteinuria and 41.5% had overt proteinuria. There were 13.9%, 24.8% and 58.5% of

patients who had CKD(stage 3a-5) in spot UPCR category of less than 0.15, 0.15 to 0.5 and more than 0.5 respectively. As the proteinuria increased quantitatively the proportion of patients who had CKD (stage 3a-5) also increased, which was statistically significant (pvalue=0.000). There is significant association between proteinuria and different stages of CKD.

C) Prevalence of CKD stages based on eGFR

Table 7: Prevalence of various stages of CKD based on eGFR

CKD Stages-G (eGFRml/min/1.73m^2)	**Numberof Patients (n)**	**Percentage(%)**
G1 (≥90)	111	22.2
G2 (60-89)	276	55.2
G3a (45-59)	66	13.2
G3b (30-44)	19	3.8
G4 (15-29)	21	4.2
G5 (<15)	7	1.4

Table7; shows the prevalence of various stages of CKD among the study population, 22.2% (n= 111) of the study participants were in stage1(G1), 55.2% (n=276) were in stage 2 (G2), 13.2% (n=66) were in stage3a(G3a), 3.8% (n=19) were in Stage 3b(G3b), 4.2% were in stage4(G4)category and 1.4% (n=7) had stage 5(G5) or End stage renal disease(ESRD). The prevalence of CKD (eGFR of < 60ml/min/1.73 m^2, ie Stage 3a -5) among the study group was 22.6%(n=113). There were 387(77.4%) study participants in CKD (stage 1-2).

D) Association of thyroid function and lipid profile with different stages of CKD.

D-1) Prevalence of Thyroid Status:

Among the study population 406(81.2%) were euthyroid and 94(18.8%) had thyroid dysfunction. Among the 94 thyroid dysfunction patients, 51 had subclinical hypothyroidism, 38 had overt hypothyroidism, 3 had subclinical hyperthyroidism and 2 had overt hyperthyroidism.

Table 8: Prevalence of Thyroid status among the different stages of CKD

Thyroid status	**CKD stage 1-2 n(%)**	**CKD stage 3a-5 n(%)**
Euthyroidism	318(82.2)	88(77.9)
Subclinical hypothyroidism	39(10.1)	12(10.6)
Overt hypothyroidism	28(6.5)	13(11.5)
Subclinicalhyperthyroidism	3(0.77)	Nil
Overthyperthyroidism	2(0.51)	Nil
Total	387(100)	113(100)

Table 8, shows the prevalence of thyroid status among different stages of CKD.AmongCKD (stage 1-2) patients 318(82.2%) wereeuthyroid and in the CKD (stage 3a-5) patients 88(77.9%) were euthyroid.Among the CKD (stage1-2) participants 39(10.1%) had subclinical hypothyroidism and among CKD (stage 3a-5) patients 12(10.6%) had subclinical hypothyroidism.Among the CKD (stage 1-2) study individuals 25(6.5%) had overt hypothyroidism, in the CKD(stage3a-5)patients,13(11.5%) had overt hypothyroidism.Among the CKD (stage1-2) 3 patients had subclinical hyperthyroidism and overt hyperthyroidism was found among 2 patients and none of the CKD (stage 3a-5) patients hadhyperthyroidism.

D-2) Table 9: Association between Thyroid status and different stages of CKD

Thyroid Status	Total n	CKD stage 1-2 n(%)	CKD stage 3a-5 n(%)	Chi-Square value	pvalue
Euthyroidism	406	318(78.3)	88(21.7)	4.612	0.202
Subclinical Hypothyroidism	51	39(76.5%)	12(23.5)		
Hypothyroidism	38	25(65.8)	13(34.2)		
Hyperthyroidism	5	5(100)	0		
Total	500	387(77.4)	113(22.6)		

Table 9, shows the association between thyroid status and different stages of CKD. Among the 51, subclinical thyroid patients 39(76.5%) had CKD (stage1-2) and 12(23.5%) of them had CKD (stage3a-5). Among the 38 hypothyroid patients, 25(65.8%) had CKD(stage 1-2) and 13(34.2%) patients had CKD(stage 3a-5). There were only 5 patients with hyperthyroidism and they had CKD(stage 1-2). There was no significant association between thyroid dysfunction and different stages of CKD (p value 0.202)

D-3) Prevalence of Dyslipidemia:

Among the study population 98(19.6%) had hypercholesterolemia,121(24.2%) had hypertriglyceridemia, 272(54.4%) had low HDLc and 69(13.8%) had high level of LDLc.

Table 10: Prevalence of dyslipidemia among different stages of CKD

Lipid profile	CKD Stage 1-2			CKD stage3a-5		
	Normal n(%)	**Abnormal n(%)**	**Total n(%)**	**Normal n(%)**	**Abnormal n(%)**	**Total n(%)**
TC	309(79.8)	**78(20.2)**	387(100)	93(82.3)	**20(17.7)**	113(100)
TGL	305(78.8)	**32(21.2)**	387(100)	74(65.5)	**39(34.5)**	113(100)
HDLc	190(49.1)	**197(50.9)**	387(100)	38(33.6)	**75(66.4)**	113(100)
LDLc	329(85)	**58(15)**	387(100)	102(90.3)	**11(9.7)**	113(100)

Table 10,shows the prevalence of dyslipidemia among the different stages of CKD.Among the CKD (stage 1-2) individuals 78(20.2%) had hypercholesterolemia and among the CKD (stage 3a-5) patients 20 (17.7%) had hypercholesterolemia.

Among the CKD (stage1-2) subjects 32(21.2%)had hypertriglyceridemia and among the CKD (stage 3a-5) patients, 39(34.5%) had hypertriglyceridemia.

Among the CKD(stage1-2) persons 197(50.9%) had low HDLc and among the CKD(stage3a-5) patients 75(66.4%) had low HDLc.

Among the CKD (stage1-2) subjects 58(15%) had high LDLc and in the CKD (stage3a-5) patients 11(9.7%) had a raised LDLc.

D-4) Table 11: Association between Dyslipidemia and different stages of CKD

Parameter	Status	CKD stage 1-2 n(%)	CKD stage3a-5 n(%)	Total n(%)	Gross Total n(%)	Chi-square value	pvalue
TC	High	78(79.6)	20(20.4)	98(19.6)			
	Normal	309(76.9)	93(23.1)	402(80.4)	500(100)	0.335	0.563
TGL	High	82(67.8)	39(32.3)	121(24.2)			
	Normal	305(80.5)	74(19.5)	379(75.8)	500(100)	8.465	**0.004***
HDLc	Low	197(72.4)	75(27.6)	272(54.4)			
	Normal	190(83.3)	38(16.7)	228(45.6)	500(100)	8.435	**0.004***
LDLc	High	58(84.1)	11(15.9)	69(13.8)			
	Normal	329(76.3)	102(23.7)	431(86.2)	500(100)	2.029	0.154

* p value < 0.05 is considered significant

Table11, shows the association between Lipid profile and different stages of CKD.Among the 98(19.6%) study subjects with raised total cholesterol levels, 78 (79.6%) had CKD (stage1-2) and 20 (20.4%) patients had CKD(stage3a-5). Among the 402(80.4%) participants with normal total cholesterol levels 76.9% had CKD(stage1-2) and 23.1% had CKD(stage3a-5). There was no significant association between total cholesterol and different stages of CKD (p value =0.563).

The Triglyceride level was raised in 121(24.2%) of the study subjects, among them 82(67.8%) had CKD (stage1-2) and 39(32.3%) patients had CKD(stage3a-5). There were 379(75.8%) subjects with normal triglyceridemia, among them 80.5% had CKD (stage 1-2) and 19.5% hadCKD(stage3a-5). There was significant association between hypertriglyceridemia and different stages of CKD (p value= 0.004).

Among the 272(54.4%) individuals who had low HDLc level, 197 (72.4%) had

CKD(stage 1-2) and 75(27.6%) of the patients had CKD(stage3a-5).Among the 228(45.6%)participants with normal HDLc level, 83.3% had CKD (stage1-2) and 16.7% hadCKD(stage3a-5). There was significant association between low HDLc and different stages of CKD (p value 0.004).

There were 69(13.8%) patients with raised LDLc level, among them 58(84.1%) had CKD (stage 1-2 and 11(15.9%) patients had CKD(stage3a-5).Among the 431(86.2%) of patients with normal LDLc level 76.3% had CKD(stage1-2) and23.7% had CKD(stage3a-5). There was no significant association between high LDLc and different stages of CKD(p value 0.154).

E) Association of risk factors like BMI, DM, HT and CVD and different stages of CKD

E-1) Prevalence of different categories of BMI:

Among the study population 190(38%) had normal BMI, 26(5.2%) were underweight, 206(41.2%) were overweight, 78(15.6%) were obese.

Table 12: Prevalence of BMI category among different stages of CKD

BMI Category	CKDstage1-2 n(%)	CKDstage3a-5 n(%)
Normal	147(38)	43(38.1)
Under weight	19(4.9)	7(6.2)
Over weight	160(41.3)	46(40.7)
Obese	61(15.6)	17(15)
Total	387(100)	113(100)

Table 12, shows the prevalence of various BMI categories among different stages of CKD. In the CKD (stage 1-2) subjects 38% had normal BMI, 4.9% were underweight, 41.3% were overweight and 15.6% were obese and in the CKD (stage 3a-5) patients 38.1% had normal BMI, 6.2% were underweight, 40.7% were over weight.15% were obese.

E-2) Table 13: Association between BMI and different stages of CKD

BMI	CKDstage1-2 n(%)	CKDstage3a-5 n(%)	Chi-square value	pvalue
Normal	147(77.4)	43(22.6)	0.315	0.95
Underweight	19(73.1)	7(26.9)		
Overweight	160(77.8)	46(22.2)		
Obese	61(78.2)	17(21.8)		
Total	387(77.4)	113(22.6)		

Table 13, shows the association between BMI and different stages of CKD. Among the participants with normal BMI,77.4% had CKD(stage1-2), 22.6% had CKD (stage 3a-5);77.8% of overweight category had CKD(stage1-2), 22.2% of overweight category of study group had CKD(stage 3a-5). Among the obese, 78.2% had CKD (stage 1-2), 21.8% had CKD (stage 3a-5). There was no significant association between BMI and different stages of CKD (p value 0.95).

E-3) Prevalence of DM, HT and CVD:

Among the study population 179(35.8%) had DM, 217(43.4%) had HT and 29(5.8%) had CVD.

Table14:P revalence of risk factors, Diabetes mellitus, Hypertension and CVD in different stages of CKD

Risk factors	**CKD stage 1-2 n(%)**			**CKD stage3a-5**		
	Present	**Absent**	**Total**	**Present**	**Absent**	**Total**
DM	**116(30)**	271(70)	387(100)	**63(55.8)**	50(44.2)	113(100)
HT	**148(38.2)**	239(61.8)	387(100)	**69(61.1)**	44(38.9)	113(100)
CVD	**15(3.9)**	372(96.1)	387(100)	**14(12.4)**	99(87.6)	113(100)

Table 14, shows the prevalence of risk factors like DM, HT and CVD. Among the individuals in CKD (stage 1-2), 30% had DM and among the CKD (stage 3a-5) patients, 55.8% had DM.Among CKD(stage1-2) individuals, 38.2% were hypertensives and among the CKD (stage 3a-5) patients 61.1% were hypertensives.Among the subjects with CKD (stage 1-2) 3.9 % had CVD and in the patients with CKD (stage 3a-5) 12.4% had CVD.

E-4) Table15: Association between Diabetes and different stages of CKD

Diabetes	CKDstage 1-2 n(%)	CKDstage3a-5 n(%)	Chi-square value	pvalue
Present	116(64.8)	63(35.2)	25.287	**0.000***
Absent	271(84.4)	50(15.6)		
Total	387(77.4)	113(22.6)		

***pvalue < 0.05 is statistically significant

Table 15, shows the association of diabetes with different stages of CKD. Among the 179 (35.8%) diabetic patients, 116(64.8%) had CKD(stage1-2) and 63 (35.2%)had CKD(stage3a-5). Among the 321(64.2%) of study individuals without diabetes, 271(84.4%) had CKD (stage1-2) and 50(15.6%) had CKD (stage3a-5).There was significant association between diabetes and different stages of CKD (p value 0.000).

E-5) Table16: Association between Hypertension and different stages of CKD

Hypertension	CKDstage1-2 n(%)	CKDstage3a-5 n(%)	Chi-square value	pvalue
Present	148(68.2%)	69(31.8)	18.54	**0.000***
Absent	239(84.5)	44(15.5)		
Total	387(77.4)	113(22.6)		

***p value < 0.05 is statistically significant

Table 16, shows the association between hypertension and CKD. Hypertension was present in 217(43.4%) of patients. Among the hypertensive patients 148(68.2%) had CKD(stage1-2) and 69(31.8%) had CKD(stage3a-5). Among the 283(56.6%) normotensive patients 239(84.5%) hadCKD(stage1-2) and 44(15.5%) had CKD (stage 3a-5). There was significant association between hypertension and different stages of CKD (p value 0.000).

E-6) Table17: Association between CVD and different stages of CKD

Cardiovascular Disease	CKDstage1-2 n(%)	CKDstage3a-5 n(%)	Chi-square value	p value
Present	15(51.7)	14(48.3)	11.602	**0.001***
Absent	372(79)	99(21.0)		
Total	387(77.4)	113(22.6)		

***p**value < 0.05 is statistically significant

Table 17, shows the association between patients with CVD and different stages of CKD. Out of 29 (5.8%) patients who had Cardio vascular disease, 15(51.7%) had CKD(stage1-2) and 14(48.3%) had CKD(stage3a-5). Among the 471(94.2%) study individuals without CVD, 372(79%) hadCKD(stage1-2) and 99(21%) had CKD(stage 3a-5).There was significant association between patients with CVD and different stages of CKD (p value 0.001).

DISCUSSION

The present study was conducted to assess the risk factors for renal dysfunction in a heterogeneous study population. The study was conducted to stress the need for early identification of risk factors causing CKD. The use of eGFR for CKD classification in this study will help in early diagnosis of CKD, because serum creatinine measurement alone will not determine the patients with stage 2 CKD. If CKD diagnosis is made early and treated, the progression of CKD can be prevented.

The prevalence of CKD (Stage 3a-5) based on the EPI eGFR (<60 ml/min/1.73m^2) in this study was 22.6%. The prevalence of different stages of CKD in this study were, stage1- 22.2%, stage2 - 55.2%, stage3a - 13.2%, stage3b - 3.8%, stage4 -4.2% and stage5-1.4%. In the Screening and Early Evaluation of Kidney disease (SEEK) study, Ajay.K.Singh et al[2] have done a population based study, among 5588 urban participants and have observed the prevalence as 17.2% (Stage 1-5) and only 6% in CKD(stages3a-5), by using CKD-EPI formula for eGFR estimation. In another study by Narinder.P.Singh et al[233]. which had 5252 semi urban participants, the prevalence among CKD (Stage 3a-5) was 13.3%, eGFR was calculated using Cockcroft-Gault formula.A study done among the apparently healthy adult central government employees, among 3398 participants, in Agra by Varma et al.[234]the prevalence of CKD(stage1-3) was 13.12% (CKD-EPI). Sanjay Kumar Agarwal et al.[235] used serum creatinine more than 1.8 mg/dl on two occasions, 8-12 weeks apart as criteria for CKD diagnosis, and has found a prevalence of CKD(Stage3-5) as 0.785%. A US study done by Vassalotti et al,[236] the Kidney Early Evaluation Programme(KEEP) the prevalence of CKD was 28.7%.Compared to other studies conducted among Indian population, the present study showed a high prevalence of CKD. All these four Indian studies were population based study and in

our study, more of diseased population is assessed and this may be attributed to the high prevalence of CKD. The very high prevalence of stage 3a-5 CKD, raises the possibility of CKD hot spot in the belt of rural and urban area of Trichy. To rule in and rule out the possible hot spot of CKD, a population based study should be performed in this geographical belt.

In this study 13% of the study population had overt proteinuria (Urine spot protein creatinine ratio more than 0.5), 26.6% had moderate proteinuria (UPCR 0.15 to 0.5). There was significant association between the proteinuria and CKD stages (p value 0.000). Many studies have found association between proteinuria and CKD. Inaguma Daijo et al.[237] in their recent study has found significant association between proteinuria and CKD and has identified proteinuria as a risk factor with CKD progressing to ESRD. KDIGO 2012[36] guidelines recommend urinary ACR as a better marker of diagnosis and progression of CKD, mainly in diabetic and hypertensive people and urinary PCR comes in the next order of preference. In this study we have analysed urinary spot PCR. Tracy Ying et al.[238] in a single centered prospective study of measuring proteinuria as the risk of progression of CKD has postulated that spot UPCR is equally good as the gold standard 24 hours proteinuria.

In this study there were 94 patients with thyroid dysfunction. Among the 500 study population, subclinical hypothyroidism was present among 10.2% of people, which was more than overt hypothyroidism (7. 6%).There were 5 (1%) hyperthyroid patients. There was no significant association between thyroid dysfunction and different stages of CKD in our study (p value0.202). In a large cross sectional study from a voluntary health examination program Yi-Cheng Chang et al.,[239] have documented a decrease in eGFR of 87.99, 83.46 and 72.22 ml/min/1.73 m^2 for euthyroid, sub clinical and overt hypothyroidism patients respectively. Similarly,

Saroj Kathwada et al.[240] had found that hypothyroidism is associated with CKD progression. Xiaolin Huang et al.[19] in their prospective study had concluded that a high serum FT4 level is associated with increased risk of CKD and they have also found no association between T3 and TSH and incident CKD. In this study we had only 5 patients with hyperthyroidism and none of them had CKD(stage3a-5). Several studies have shown that there is increase in eGFR in hyperthyroidism. In hyperthyroidism an increase of 17-18% of eGFR has been noticed and this rise in GFR is due to increase in renal blood flow and activation of renin angiotensin aldosterone system.[118] Hyperthyroidism causes CKD by increasing the renal blood flow leading to intra glomerular hypertension and increased filtration pressure. Associated proteinuria in hyperthyroidism also causes renal injury.[241]

In our study the relationship between dyslipidemia and different stages CKD was assessed, serum levels of total cholesterol, HDLc, TGLc and LDLc were analysed and their relationship with CKD was studied. Among the study population 19.6% had hypercholesterolemia, 24.2% had high triglyceride level, 54.4% had low HDLc and 13.8% had high LDLc levels. There was significant association between CKD and high TGL levels (pvalue= 0.004) and low HDLc (p value=0.004). But there was no significant association between CKD and high levels of totalcholesterol (p value=0.563) and LDLc(pvalue=0.154).Saroj Khatiwada et al[240] in their cross sectional study, conducted among 360 CKD patients have stated that hypercholesterolemia, low HDLc, hypertriglyceridemia and undesirable LDL cholesterol were significantly associated with presence of cardiovascular disease in CKD. But did not find significant rise in dyslipidemia cases during CKD progression. Our study also did not show significant association for total cholesterol and LDLc and CKD. Zhongshang Yuan et al[102] in a large scale multicentric cross-sectional study

(National community based program) has concluded that TG/HDL ratio was associated with renal dysfunction and also exhibited a significantly strong association between increase in serum creatinine and CKD in hypothyroidism and euthyroidism.

In this study, 38% the study subjects had normal BMI, 41.2% were overweight and 15.6% were obese. There were more participants in the overweight category. There was no significant association between obesity and CKD in our study (pvalue 0.95). A study done by Zaman et al.,[242]has shown a negative association between BMI and CKD in a study done among Type 2 DM. Similarly, Narinder P Singh et al.[233] has found an inverse association between BMI and renal impairment. The BMI assess both the central and peripheral fat, muscle mass and fluid. As it is the central obesity that determines the cardiovascular and the renal risk, BMI might not have accurately shown the association with CKD. A lot of studies have stated obesity as an important risk factor for CKD. Christiana Aryee et al.[144] had stated that in their study higher anthropometric were recorded among hypertensive nephropathy patients.Andrzej Jaroszynski et al.[243] had demonstrated that central obesity (waist circumference and waist hip ratio) showed higher discriminative ability for CKD than BMI. In this study we found a significant difference between the mean values of waist circumference between the CKD (stage1-2) and CKD (stage 3a-5) (p value=0.02). In early stages of obesity there will be increased GFR. The mechanism for early rise in GFR in obesity may be due to macula densa feedback mechanism, the visceral obesity causes compression of kidneys which may cause an increase in sodium reabsorption in loop of Henle hence less sodium in the macula densa and causes a tubulo glomerular feedback and increases renin secretion, increased blood flow and GFR. The glomerular hyper filtration then gradually subsides and there will be a gradual decrease in GFR.[153]

The prevalence of Diabetes mellitus among our study population was 35.8%, and among the CKD patients 47.8% had diabetics. There was significant association between diabetes and CKD stages (p value 0.000). Diabetes is the second most common risk factor for CKD in our study. In a study done by Wu Bingcao et al.[244] the prevalence of CKD in T2DM was 38.3%, which is similar to that of our study.The study by Justin Gatwood et al,[245]also had reported a prevalence of 40%, which is nearly similar to our study .In a retrospective hospital based study done in Assam, Manjuri Sharma et al,[246]have identified diabetes mellitus(44.2%) as a leading cause of CKD. Sajith Kumar Soman et al,[247] have concluded in their study that CKD was highly prevalent among diabetics and the duration of diabetes, urinary protein levels and RBS value had a strong association with CKD in diabetes.

The prevalence of Hypertension was 43.4% and the prevalence of hypertension among CKD (stage 3a-5), patients was 61.1% and there was significant association between hypertension and different stages of CKD (p value=0.000). In our study hypertension was the most common risk factor for CKD. A lot of studies have stated hypertension as a common risk factor for CKD. The SEEK study done by Ajay.K.Singh et al.[2]showed a high prevalence of hypertension(65%) and identified hypertension as one of the most common risk factor for CKD. Kayori Hayashi et al[5], in their study of metabolic factors associated with CKD have found that hypertension was associated with rapid decline in eGFR and hence need to be monitored for prevention of occurrence of CKD. Narinder.P.Singh et al[233]. in a population based study have found a CKD prevalence of 17.1% among hypertensives and have stated that hypertension plays an important role as risk factor in CKD development.

In our study population Cardiovascular disease(CVD) was found in 29 (5.8%) patients. Among the CKD (stage 3a-5) patients in our study 12.4% had CVD. There

was a significant association between CVD and different stages of CKD (p value=0.001). Several studies have shown association between CKD and CVD. Luis .M.Ruilope et al[248] in their HOT study has stated that patients who had renal insufficiency as identified by a baseline creatinine of >1.5 mg/dl or eGFR< 60 ml/min/1.73 m^2 had an increased risk of CVD. In the HOPE randomized trial Johannes.F.E.Mann et al,[249] have concluded that in patients with pre-existing vascular disease or diabetes combined with cardiovascular risk, mild renal insufficiency significantly increased the risk of CVD. Bruce. F. Culleton et al.[250] in a community based cohort have found that mild renal insufficiency is common in the community and those with mild renal insufficiency were associated with high prevalence of CVD. This may be because many traditional CVD risk factors like hypertension, hypertriglyceridemia low HDLc etc. are themselves risk factors for CKD.

SUMMARY

In this cross-sectional study among a heterogeneous adult population in a tertiary care centre, 500 adults of both sexes were evaluated for their renal status. The study showed a prevalence of Chronic Kidney Disease (stage 3a-5) (based on eGFR < 60ml/min/1.73m^2) to be 22.6% and the prevalence of CKD stage 2 was 55.2%. With increase in age there was a significant decrease in eGFR. In this study male sex had a higher prevalence of CKD (stage 3a-5), as the age increased. In this study 60.4% of the study subjects had normal proteinuria and 13% had overt proteinuria. There was significant association between proteinuria and different stages of CKD.

Sub clinical hypothyroidism was the prevalent (10.2%) thyroid dysfunction, among the study population. There was no significant association between thyroid dysfunction and different stages of CKD.On study of association between dyslipidemia and different stages of CKD, significant association was found between low HDLc and high TGL levels and different CKD stages, whereas increased levels of total cholesterol and high LDLc levels were not significantly associated with CKD.

Among the conventional risk factors like obesity, HT and DM, in the study of association of different BMI category and different stages of CKD, 41.2% of the study population were overweight. Obesity by BMI was not significantly associated with different CKD stages, but central obesity, by waist circumference, the mean values showed a significant difference between the different CKD stages (p value 0.02). The prevalence of hypertension was high (43.4%) in the study population. Among the patients with CKD (stage 3a-5) 61.1% were hypertensives. The prevalence of diabetes mellitus among the study population was 35.8% and 47.8% of CKD (stage 3a-5) were diabetics. Hypertension was the most common risk factor for CKD in the study population. CKD was significantly associated with the common risk

factors like hypertension and diabetes.

On studying the association between CVD and different stages of CKD, 12.4% of the CKD (stage 3a-5) had CVD. Cardiovascular disease was also significantly associated with different stages of CKD.

Based on the study done, it is suggested that estimated Glomerular Filtration Rate (eGFR) may be included in the laboratory reports of all patients with risk factors like Diabetes Mellitus, Hypertension and Obesity for early identification of renal dysfunction. Patients with Cardiovascular disease and dyslipidemia may be investigated for renal dysfunction.

CONCLUSION

- The prevalence of Chronic Kidney Disease was high in the study population.
- The prevalence of CKD was more among males than females in our study population.
- Hypertension, Diabetes mellitus, Cardiovascular disease and Proteinuria were associated significantly with different stages of CKD.
- Hypertension was the most common risk factor among CKD patients.
- There was significant association between high triglyceride levels and low HDLc levels and different stages of CKD.

LIMITATION OF THE STUDY

- As this was an observational study the progression pattern of CKD could not be assessed.
- This study was conducted among hospital population which represents a diseased population rather than a representation of general population.

REFERENCES

1. Levin A, Stevens PE. Summary of KDIGO 2012 CKD Guideline: behind the scenes, need for guidance, and a framework for moving forward. Kidney Int. 2014 Jan;85(1):49–61.

2. Singh AK, Farag YM, Mittal BV, Subramanian KK, Reddy SR, Acharya VN, Almeida AF, Channakeshavamurthy A, Ballal HS, Gaccione P, Issacs R. Epidemiology and risk factors of chronic kidney disease in India–results from the SEEK (Screening and Early Evaluation of Kidney Disease) study. BMC nephrology. 2013 Dec;14(1):114.

3. Coresh J, Hu J-R, Bello AK, Feldman HI, Fogo AB, Ganji MR, et al. Action plan for determining and monitoring the prevalence of chronic kidney disease. Kidney Int Suppl. 2017 Oct 1;7(2):63–70.

4. Raines N, González M, Wyatt C, Kurzrok M, Pool C, Lemma T, et al. Risk factors for reduced glomerular filtration rate in a Nicaraguan community affected by mesoamerican nephropathy. MEDICC Rev. 2014 Apr;16:16–22.

5. Hayashi K, Takayama M, Abe T, Kanda T, Hirose H, Shimizu-Hirota R, et al. Investigation of Metabolic Factors Associated with eGFR Decline Over 1 Year in a Japanese Population without CKD. J Atheroscler Thromb. 2017 Aug 1;24(8):863–75.

6. Adrogué HJ, Madias NE. Sodium and Potassium in the Pathogenesis of Hypertension. N Engl J Med. 2007 May 10;356(19):1966–78.

7. Drenjančević-Perić I, Jelaković B, Lombard JH, Kunert MP, Kibel A, Gros M. High-Salt Diet and Hypertension: Focus on the Renin-Angiotensin System. Kidney Blood Press Res. 2011;34(1):1–11.

8. Zhou X, Matavelli L, Frohlich ED. Uric acid: Its relationship to renal hemodynamics and the renal renin-angiotensin system. Curr Hypertens Rep. 2006 Mar 1;8(2):120–4.

9. Kang H-T, Shim J-Y, Lee Y-J, Lee J-E, Linton JA, Kim J-K, et al. Association between the Ratio of Triglycerides to High-Density Lipoprotein Cholesterol and Chronic Kidney Disease in Korean Adults: The 2005 Korean National Health and Nutrition Examination Survey. Kidney Blood Press Res. 2011;34(3):173–9.

10. Ritz E, Wanner C. Lipid Changes and Statins in Chronic Renal Insufficiency. J Am Soc Nephrol. 2006 Dec 1;17(12 suppl 3):S226–30.

11. Parikh NI, Hwang SJ, Larson MG, Meigs JB, Levy D, Fox CS. Cardiovascular disease risk factors in chronic kidney disease: overall burden and rates of treatment and control. Archives of internal medicine. 2006 Sep 25;166(17):1884-91.

12. Kim J-Y, Kang H-T, Lee H-R, Lee Y-J, Shim J-Y. Comparison of Lipid-Related Ratios for Prediction of Chronic Kidney Disease Stage 3 or More in Korean Adults. J Korean Med Sci. 2012;27(12):1524.

13. Johnson ALICM, Stahl A, Zager RA. Triglyceride accumulation in injured renal tubular cells: Alterations in both synthetic and catabolic pathways. Kidney Int. 2005 Jun;67(6):2196–209.

14. Baragetti A, Norata GD, Sarcina C, Rastelli F, Grigore L, Garlaschelli K, Uboldi P, Baragetti I, Pozzi C, Catapano AL. High density lipoprotein cholesterol levels are an independent predictor of the progression of chronic kidney disease. Journal of internal medicine. 2013 Sep;274(3):252-62.

15. Sun K, Lin D, Li F, Huang C, Qi Y, Xue S, Tang J, Yang C, Li Y, Ren M, Yan L. Discordant associations of lipid parameters with albuminuria and chronic kidney disease: a population-based study. Lipids in health and disease. 2015 Dec;14(1):152.

16. Villabona C, Sahun M, Gómez N, Gómez JM, Soler J, Roca M, et al. Blood Volumes and Renal Function in Overt and Subclinical Primary Hypothyroidism. Am J Med Sci. 1999 Oct 1;318(4):277–80.

17. Hollander JGD, Wulkan RW, Mantel MJ, Berghout A. Correlation between severity of thyroid dysfunction and renal function. Clin Endocrinol (Oxf). 62(4):423–7.

18. Zhang Y, Chang Y, Ryu S, Cho J, Lee W-Y, Rhee E-J, et al. Thyroid hormone levels and incident chronic kidney disease in euthyroid individuals: the Kangbuk Samsung Health Study. Int J Epidemiol. 2014 Oct 1;43(5):1624–32.

19. Huang X, Ding L, Peng K, Lin L, Wang T, Zhao Z, Xu Y, Lu J, Chen Y, Wang W, Bi Y. Thyroid hormones associate with risk of incident chronic kidney disease and rapid decline in renal function: a prospective investigation. Journal of translational medicine. 2016 Dec;14(1):336.

20. Cooper DS, Biondi B. Subclinical thyroid disease. The Lancet. 2012 Mar 24;379(9821):1142–54.

21. Åsvold BO, Bjøro T, Vatten LJ. Association of thyroid function with estimated glomerular filtration rate in a population-based study: the HUNT study. Eur J Endocrinol. 2011 Jan 1;164(1):101–5.

22. Canaris GJ, Manowitz NR, Mayor G, Ridgway EC. The Colorado thyroid disease prevalence study. Archives of internal medicine. 2000 Feb 28;160(4):526-34

23. García-Trabanino R, Jarquín E, Wesseling C, Johnson RJ, González-Quiroz M, Weiss I, et al. Heat stress, dehydration, and kidney function in sugarcane cutters in El Salvador – A cross-shift study of workers at risk of Mesoamerican nephropathy. Environ Res. 2015 Oct 1;142:746–55.

24. Soderland P, Lovekar S, Weiner DE, Brooks DR, Kaufman JS. Chronic Kidney Disease Associated With Environmental Toxins and Exposures. Adv Chronic Kidney Dis. 2010 May 1;17(3):254–64.

25. McClean M, Laws R, Rubio OR, Brooks D, Kaufman J, Weiner D, Nicholson S, Miller A, Makey C, Collins E, Ames J. Industrial hygiene/occupational health assessment: evaluating potential hazards associated with chemicals and work practices at the Ingenio San Antonio (Chichigalpa, Nicaragua). Boston, MA: Boston University School of Public Health. 2010.

26. Ting SMS, Nair H, Ching I, Taheri S, Dasgupta I. Overweight, Obesity and Chronic Kidney Disease. Nephron Clin Pract. 2009;112(3):c121–7.

27. Burton JO, Gray LJ, Webb DR, Davies MJ, Khunti K, Crasto W, et al. Association of anthropometric obesity measures with chronic kidney disease risk in a non-diabetic patient population. Nephrol Dial Transplant. 2012 May 1;27(5):1860–6.

28. Haroun MK, Jaar BG, Hoffman SC, Comstock GW, Klag MJ, Coresh J. Risk Factors for Chronic Kidney Disease: A Prospective Study of 23,534 Men and Women in Washington County, Maryland. J Am Soc Nephrol. 2003 Nov 1;14(11):2934–41.

29. Orth SR, Hallan SI. Smoking: A Risk Factor for Progression of Chronic Kidney Disease and for Cardiovascular Morbidity and Mortality in Renal Patients—Absence of Evidence or Evidence of Absence? Clin J Am Soc Nephrol. 2008 Jan 1;3(1):226–36.

30. Hall JE. Guyton and Hall Textbook of Medical Physiology E-Book. Elsevier Health Sciences; 2015. 1172 p.

31. Delaney.P.Michael,Price.P.Christopher,Lamb.J.Edmund.Tietz Textbook of Clinical Chemistry and Molecular Diagnostics.5th ed. Philadelphia.Elsevier.2012.48;Kidney Disease;p.1540-41.

32. Rüster C, Wolf G. Renin-Angiotensin-Aldosterone System and Progression of Renal Disease. J Am Soc Nephrol. 2006 Nov 1;17(11):2985–91.

33. Zatz R. Haemodynamically mediated glomerular injury: the end of a 15-year-old controversy? Curr Opin Nephrol Hypertens. 1996 Nov;5(6):468.

34. Remuzzi G, Ruggenenti P, Benigni A. Understanding the nature of renal disease progression. Kidney Int. 1997 Jan;51(1):2–15.

35. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease.Kidney International Supplement.2013;3(1):5

36. Inker LA, Astor BC, Fox CH, Isakova T, Lash JP, Peralta CA, et al. KDOQI US Commentary on the 2012 KDIGO Clinical Practice Guideline for the Evaluation and Management of CKD. Am J Kidney Dis. 2014 May;63(5):713–35.

37. Levin A, Stevens PE. Summary of KDIGO 2012 CKD Guideline: behind the scenes, need for guidance, and a framework for moving forward. Kidney Int. 2014 Jan;85(1):49–61.

38. Myers GL. Recommendations for Improving Serum Creatinine Measurement: A Report from the Laboratory Working Group of the National Kidney Disease Education Program. Clin Chem. 2006 Jan 1;52(1):5–18.

39. Levey AS, Coresh J, Bolton K, Culleton B, Harvey KS, Ikizler TA, Johnson CA, Kausz A, Kimmel PL, Kusek J, Levin A. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. American Journal of Kidney Diseases. 2002;39(2 SUPPL. 1)

40. Imai E, Matsuo S, Makino H, Watanabe T, Akizawa T, Nitta K, Iimuro S, Ohashi Y, Hishida A. Chronic Kidney Disease Japan Cohort study: baseline characteristics and factors associated with causative diseases and renal function. Clinical and experimental nephrology. 2010 Dec 1;14(6):558-70.

41. Luyckx VA, Tuttle KR, Garcia-Garcia G, Gharbi MB, Heerspink HJL, Johnson DW, et al. Reducing major risk factors for chronic kidney disease. Kidney Int Suppl. 2017 Oct;7(2):71–87.

42. Cases A, Coll E. Dyslipidemia and the progression of renal disease in chronic renal failure patients. Kidney Int. 2005 Dec;68:S87–93.

43. Senthilnathan G, Prabhakaran R. A Study of Evaluation of Thyroid Function Status In Patients With Chronic Kidney Disease. :4.

44. Perrone RD, Madias NE, Levey AS. Serum Creatinine as an Index of Renal Function: New Insights into Old Concepts. :21.

45. Florkowski CM, Chew-Harris JS. Methods of Estimating GFR – Different Equations Including CKD-EPI. Clin Biochem Rev. 2011 May;32(2):75–9.

46. Cockcroft DW, Gault H. Prediction of Creatinine Clearance from Serum Creatinine. Nephron. 1976;16(1):31–41.

47. Levey AS. A More Accurate Method To Estimate Glomerular Filtration Rate from Serum Creatinine: A New Prediction Equation. Ann Intern Med. 1999 Mar 16;130(6):461.

48. Lin J, Knight EL, Hogan ML, Singh AK. A Comparison of Prediction Equations for Estimating Glomerular Filtration Rate in Adults without Kidney Disease. J Am Soc Nephrol. 2003 Oct 1;14(10):2573–80.

49. Levey AS, Stevens LA, Schmid CH, Zhang Y (Lucy), Castro AF, Feldman HI, et al. A New Equation to Estimate Glomerular Filtration Rate. Ann Intern Med. 2009 May 5;150(9):604.

50. Roos JF, Doust J, Tett SE, Kirkpatrick CMJ. Diagnostic accuracy of cystatin C compared to serum creatinine for the estimation of renal dysfunction in adults and children—A meta-analysis. Clin Biochem. 2007 Mar 1;40(5):383–91.

51. Dharnidharka VR, Kwon C, Stevens G. Serum cystatin C is superior to serum creatinine as a marker of kidney function: A meta-analysis. Am J Kidney Dis. 2002 Aug 1;40(2):221–6.

52. Schwartz GJ, Work DF. Measurement and Estimation of GFR in Children and Adolescents. Clin J Am Soc Nephrol. 2009 Nov 1;4(11):1832–43.

53. Schwartz GJ, Muñoz A, Schneider MF, Mak RH, Kaskel F, Warady BA, et al. New Equations to Estimate GFR in Children with CKD. J Am Soc Nephrol JASN. 2009 Mar;20(3):629–37.

54. Price CP. Use of Protein:Creatinine Ratio Measurements on Random Urine Samples for Prediction of Significant Proteinuria: A Systematic Review. Clin Chem. 2005 Jul 14;51(9):1577–86.

55. Katayev A, Zebelman AM, Sharp TM, Samantha Flynn, Bernstein RK. Prevalence of isolated non-albumin proteinuria in the US population tested for both, urine total protein and urine albumin: An unexpected discovery. Clin Biochem. 2017 Apr 1;50(6):262–9.

56. Barratt J, Topham P. Urine proteomics: the present and future of measuring urinary protein components in disease. Can Med Assoc J. 2007 Aug 14;177(4):361–8.

57. Ginsberg JM, Chang BS, Matarese RA, Garella S. Use of Single Voided Urine Samples to Estimate Quantitative Proteinuria. N Engl J Med. 1983 Dec 22;309(25):1543–6.

58. Jong PE de, Curhan GC. Screening, Monitoring, and Treatment of Albuminuria: Public Health Perspectives. J Am Soc Nephrol. 2006 Aug 1;17(8):2120–6.

59. Cieciura T, Urbanowicz A, Perkowska-Ptasinska A, Nowacka-Cieciura E, Tronina O, Majchrzak J, et al. Tubular and Glomerular Proteinuria in Diagnosing Chronic Allograft Nephropathy With Relevance to the Degree of Urinary Albumin Excretion. Transplant Proc. 2005 Mar 1;37(2):987–90.

60. Halimi J-M, Matthias B, Al-Najjar A, Laouad I, Chatelet V, Marlière J-F, et al. Respective Predictive Role of Urinary Albumin Excretion and Nonalbumin Proteinuria on Graft Loss and Death in Renal Transplant Recipients. Am J Transplant. 7(12):2775–81.

61. Kim JH, Oh SY, Kim EH, Lee MJ, Jeon YK, Kim BH, et al. Addition of nonalbumin proteinuria to albuminuria improves prediction of type 2 diabetic nephropathy progression. Diabetol Metab Syndr. 2017 Sep 6;9:68.

62. Brunskill NJ. Albumin handling by proximal tubular cells: mechanisms and mediators. Nephrology Dialysis Transplantation. 2000 Dec 1;15(90006):39-40

63. Eddy AA, Giachelli CM, McCulloch with the technical assistance of L, Liu E. Renal expression of genes that promote interstitial inflammation and fibrosis in rats with protein-overload proteinuria. Kidney Int. 1995 Jun 1;47(6):1546–57.

64. Powers.C.Alvin.Harrison's Principles of Internal Medicine 19th ed.(Vol.2).United States of America. Mc Graw Hill education.2015.419; Diabetes Mellitus Complication;p.2424

65. Ziyadeh FN. The Extracellular Matrix in Diabetic Nephropathy. Am J Kidney Dis. 1993 Nov 1;22(5):736–44.

66. Ziyadeh FN. Mediators of hyperglycemia and the pathogenesis of matrix accumulation in diabetic renal disease. Miner Electrolyte Metab. 1995;21(4–5):292–302.

67. Bleyer AJ, Fumo P, Snipes ER, Goldfarb S, Simmons DA, Ziyadeh FN. Polyol pathway mediates high glucose-induced collagen synthesis in proximal tubule. Kidney Int. 1994 Mar;45(3):659–66.

68. Goldfarb S, Ziyadeh FN, Kern EFO, Simmons DA. Effects of Polyol-Pathway Inhibition and Dietary myo-Inositol on Glomerular Hemodynamic Function in Experimental Diabetes Mellitus in Rats. Diabetes. 1991 Apr 1;40(4):465–71.

69. Studer RK, Craven PA, DeRubertis FR. Role for Protein Kinase C in the Mediation of Increased Fibronectin Accumulation by Mesangial Cells Grown in High-Glucose Medium. Diabetes. 1993 Jan 1;42(1):118–26.

70. Fumo P, Kuncio GS, Ziyadeh FN. PKC and high glucose stimulate collagen alpha 1 (IV) transcriptional activity in a reporter mesangial cell line. Am J Physiol-Ren Physiol. 1994 Oct 1;267(4):F632–8.

71. Cohen MP, Sharma K, Jin Y, Hud E, Wu VY, Tomaszewski J, et al. Prevention of diabetic nephropathy in db/db mice with glycated albumin antagonists. A novel treatment strategy. J Clin Invest. 1995 May 1;95(5):2338–45.

72. Brownlee M M. Advanced Protein Glycosylation in Diabetes and Aging. Annu Rev Med. 1995;46(1):223–34.

73. Sharma K, Ziyadeh FN. Hyperglycemia and Diabetic Kidney Disease: The Case for Transforming Growth Factor–β as a Key Mediator. Diabetes. 1995 Oct 1;44(10):1139–46.

74. Border WA, Ruoslahtit E, ofNephrology D. Transforming Growth Factor-,B in Disease: The Dark Side of Tissue Repair. :7.

75. Young BA, Johnson RJ, Alpers CE, Eng E, Gordon K, Floege J, et al. Cellular events in the evolution of experimental diabetic nephropathy. Kidney Int. 1995 Mar;47(3):935–44.

76. Inaba T, Ishibashi S, Gotoda T, Kawamura M, Morino N, Nojima Y, et al. Enhanced Expression of Platelet-Derived Growth Factor-β Receptor by High Glucose Involvement of Platelet-Derived Growth Factor in Diabetic Angiopathy. Diabetes. 1996 Apr 1;45(4):507–12.

77. Hammerman MR. `The growth hormone–insulin-like growth factor axis in kidney re-revisited. Nephrol Dial Transplant. 1999 Aug 1;14(8):1853–60.

78. Flyvbjerg A, Orskov H. Kidney tissue insulin-like growth factor I and initial renal growth in diabetic rats: Relation to severity of diabetes. Eur J Endocrinol. 1990 Mar 1;122(3):374–8.

79. Liu Y, Tolbert EM, Sun AM, Dworkin LD. In vivo and in vitro evidence for increased expression of HGF receptor in kidney of diabetic rat. Am J Physiol-Ren Physiol. 1996 Dec;271(6):F1202–10.

80. Wolf G, Mueller E, Stahl RA, Ziyadeh FN. Angiotensin II-induced hypertrophy of cultured murine proximal tubular cells is mediated by endogenous transforming growth factor-beta. The Journal of clinical investigation. 1993 Sep 1;92(3):1366-72.

81. Kennefick TM, Anderson S. Role of angiotensin II in diabetic nephropathy. Semin Nephrol. 1997 Sep;17(5):441–7.

82. Nakamura T, Ebihara I, Tomino Y, Koide H. Alteration of growth-related proto-oncogene expression in diabetic glomeruli by a specific endothelin receptor A antagonist. Nephrology Dialysis Transplantation. 1996 Aug 1;11(8):1528-31.

83. Craven PA, Patterson MC, DeRubertis FR. Role of Enhanced Arachidonate Availability Through Phospholipase A2 Pathway in Mediation of Increased Prostaglandin Synthesis by Glomeruli From Diabetic Rats. Diabetes. 1988 Apr 1;37(4):429–35.

84. Wolf G, Ziyadeh FN. Molecular mechanisms of diabetic renal hypertrophy. Kidney Int. 1999 Aug 1;56(2):393–405.

85. Reusch JEB. Diabetes, microvascular complications, and cardiovascular complications: what is it about glucose? J Clin Invest. 2003 Oct 1;112(7):986–8.

86. Haneda M, Araki S, Togawa M, Sugimoto T, Isono M, Kikkawa R. Activation of mitogen-activated protein kinase cascade in diabetic glomeruli and mesangial cells cultured under high glucose conditions. Kidney Int Suppl. 1997 Sep;60:S66-69.

87. Haneda M, Araki S, Togawa M, Sugimoto T, Motohide I, Kikkawa R. Mitogen-Activated Protein Kinase Cascade Is Activated in Glomeruli of Diabetic Rats and Glomerular Mesangial Cells Cultured Under High Glucose Conditions. Diabetes. 1997 May 1;46(5):847–53.

88. Simm A, Münch G, Seif F, Schenk O, Heidland A, Richter H, et al. Advanced glycation endproducts stimulate the MAP-kinase pathway in tubulus cell line LLC-PK1. FEBS Lett. 410(2–3):481–4.

89. Nagata S. Apoptosis by Death Factor. Cell. 1997 Feb 7;88(3):355–65.

90. Reed JC. Bcl-2 and the regulation of programmed cell death. The Journal of cell biology. 1994 Jan;124(1-2):1-6.

91. Ortiz A, Ziyadeh FN, Neilson EG. Expression of apoptosis-regulatory genes in renal proximal tubular epithelial cells exposed to high ambient glucose and in diabetic kidneys. J Investig Med Off Publ Am Fed Clin Res. 1997 Feb;45(2):50–6.

92. Vaziri ND. Dyslipidemia of chronic renal failure: the nature, mechanisms, and potential consequences. Am J Physiol-Ren Physiol. 2006 Feb;290(2):F262–72.

93. Stefanovic V, Milojkovic M. Treatment of Dyslipidemia in Chronic Kidney Disease. Int J Artif Organs. 2004 Oct 1;27(10):821–7.

94. Attman P, Knight-Gibson C, Tavella M, Samuelsson O, Alaupovic P. The compositional abnormalities of lipoproteins in diabetic renal failure. Nephrol Dial Transplant. 1998 Nov 1;13(11):2833–41.

95. Kronenberg F, Kuen E, Ritz E, Junker R, König P, Kraatz G, et al. Lipoprotein(a) Serum Concentrations and Apolipoprotein(a) Phenotypes in Mild and Moderate Renal Failure. J Am Soc Nephrol. 2000 Jan 1;11(1):105–15.

96. Weintraub M, Burstein A, Rassin T, Liron M, Ringel Y, Cabili S, et al. Severe defect in clearing postprandial chylomicron remnants in dialysis patients. Kidney Int. 1992 Nov;42(5):1247–52.

97. Attman P-O, Samuelsson O, Alaupovic P. Lipoprotein Metabolism and Renal Failure. Am J Kidney Dis. 1993 Jun 1;21(6):573–92.

98. Heuck CC, Liersch M, Ritz E, Stegmeier K, Wirth A, Mehls O. Hyperlipoproteinemia in experimental chronic renal insufficiency in the rat. Kidney Int. 1978 Aug;14(2):142–50.

99. Majumdar A. Lipid abnormalities in renal disease. 2000;93:5.

100. Shoji T, Nishizawa Y, Nishitani H, Yamakawa M, Morii H. Impaired metabolism of high density lipoprotein in uremic patients. Kidney Int. 1992 Jun;41(6):1653–61.

101. Klin M, Smogorzewski M, Ni Z, Zhang G, Massry SG. Abnormalities in hepatic lipase in chronic renal failure: role of excess parathyroid hormone. J Clin Invest. 1996 May 15;97(10):2167–73.

102. Yuan Z, Zhao M, Zhang B, Zhang H, Zhang X, Guan Q, Ning G, Gao L, Xue F, Zhao J. Thyroid function modifies the association between ratio of triglyceride to high-density lipoprotein cholesterol and renal function: a multicenter cross-sectional study. Scientific reports. 2015 Jul 16;5:11052.

103. Fujisawa M, Haramaki R, Miyazaki H, Imaizumi T, Okuda S. Role of Lipoprotein (a) and TGF-β1 in Atherosclerosis of Hemodialysis Patients. J Am Soc Nephrol. 2000 Oct 1;11(10):1889–95.

104. Moorhead JF, El-Nahas M, Chan MK, Varghese Z. LIPID NEPHROTOXICITY IN CHRONIC PROGRESSIVE GLOMERULAR AND TUBULO-INTERSTITIAL DISEASE. The Lancet. 1982 Dec 11;320(8311):1309–11.

105. Abrass CK. Cellular Lipid Metabolism and the Role of Lipids in Progressive Renal Disease. Am J Nephrol. 2004;24(1):46–53.

106. Joles JA, Kunter U, Janssen U, Kriz W, Rabelink TJ, Koomans HA, et al. Early Mechanisms of Renal Injury in Hypercholesterolemic or Hypertriglyceridemic Rats. J Am Soc Nephrol. 2000 Apr 1;11(4):669–83.

107. Sun L, Halaihel N, Zhang W, Shimomura I, Horton JD, Rogers T, Levi M. Role of SREBP-1 in regulation of renal lipid metabolism and glomerulosclerosis in diabetes mellitus. Journal of Biological Chemistry. 2002 Mar 1.

108. Jiang T, Wang Z, Proctor G, Moskowitz S, Liebman SE, Rogers T, et al. Diet-induced Obesity in C57BL/6J Mice Causes Increased Renal Lipid Accumulation and Glomerulosclerosis via a Sterol Regulatory Element-binding Protein-1c-dependent Pathway. J Biol Chem. 2005 Sep 16;280(37):32317–25.

109. van Hoek I, Daminet S. Interactions between thyroid and kidney function in pathological conditions of these organ systems: A review. Gen Comp Endocrinol. 2009 Feb 1;160(3):205–15.

110. Iglesias P, Díez JJ. Thyroid dysfunction and kidney disease. Eur J Endocrinol. 2009 Apr 1;160(4):503–15.

111. Kumar J, Gordillo R, Kaskel FJ, Druschel CM, Woroniecki RP. Increased Prevalence of Renal and Urinary Tract Anomalies in Children with Congenital Hypothyroidism. J Pediatr. 2009 Feb 1;154(2):263–6.

112. Canavan JP, Holt J, Easton J, Smith K, Goldspink DF. Thyroid-induced changes in the growth of the Liver, Kidney, and Diaphragm of Neonatal Rats. Journal of cellular physiology. 1994 Oct;161(1):49-54.

113. Lin H-H, Tang M-J. Thyroid hormone upregulates Na,K-ATPase α and β mRNA in primary cultures of proximal tubule cells. Life Sci. 1997 Jan 3;60(6):375–82.

114. Katz AI, Lindheimer MD. Renal Sodium- and Potassium-Activated Adenosine Triphosphatase and Sodium Reabsorption in the Hypothyroid Rat. J Clin Invest. 1973 Apr 1;52(4):796–804.

115. Vaamonde CA, Sebastianelli MJ, Vaamonde LS, Pellegrini EL, Watts RS, Klingler EL, et al. Impaired renal tubular reabsorption of sodium in hypothyroid man. Transl Res. 1975 Mar 1;85(3):451–66.

116. Pracyk JB, Slotkin TA. Thyroid hormone differentially regulates development of beta-adrenergic receptors, adenylate cyclase and ornithine decarboxylase in rat heart and kidney. J Dev Physiol. 1991 Oct;16(4):251–61.

117. Segarra AB, Ramírez M, Banegas I, Hermoso F, Vargas F, Vives F, et al. Influence of Thyroid Disorders on Kidney Angiotensinase Activity. Horm Metab Res. 2006 Jan;38(1):48–52.

118. Haro JM, Sabio JM, Vargas F. Renal β-adrenoceptors in thyroxine-treated rats. J Endocrinol Invest. 1992 Sep 1;15(8):605–8.

119. Baum M, Dwarakanath V, Alpern RJ, Moe OW. Effects of thyroid hormone on the neonatal renal cortical Na+/H+ antiporter. Kidney Int. 1998 May;53(5):1254–8.

120. Prisant LM, Gujral JS, Mulloy AL. Hyperthyroidism: a secondary cause of isolated systolic hypertension. J Clin Hypertens Greenwich Conn. 2006 Aug;8(8):596–9.

121. Basu G, Mohapatra A. Interactions between thyroid disorders and kidney disease. Indian J Endocrinol Metab. 2012;16(2):204–13.

122. Kreisman SH, Hennessey JV. Consistent reversible elevations of serum creatinine levels in severe hypothyroidism. Archives of internal medicine. 1999 Jan 11;159(1):79-82.

123. Mooraki A, Broumand B, Neekdoost F, Amirmokri P, Bastani B. Reversible acute renal failure associated with hypothyroidism: Report of four cases with a brief review of literature. Nephrology. 8(2):57–60.

124. Karanikas G, Schütz M, Szabo M, Becherer A, Wiesner K, Dudczak R, et al. Isotopic Renal Function Studies in Severe Hypothyroidism and after Thyroid Hormone Replacement Therapy. Am J Nephrol. 2004;24(1):41–5.

125. Al-Fifi S, Girardin C, Sharma A, Rodd C. Moderate renal failure in association with prolonged acquired hypothyroidism in children. Acta Pædiatrica. 2007 Jan 2;88(7):715–7.

126. Åsvold BO, Bjøro T, Vatten LJ. Association of thyroid function with estimated glomerular filtration rate in a population-based study: the HUNT study. Eur J Endocrinol. 2011 Jan 1;164(1):101–5.

127. Woodward A, McCann S, Al-Jubouri M. The relationship between estimated glomerular filtration rate and thyroid function: an observational study. Ann Clin Biochem. 2008 Sep 1;45(5):515–7.

128. Van MW, Lobatto S. Treatment of severe hypothyroidism in a patient with progressive renal failure leads to significant improvement of renal function. Clin Nephrol. 2007 Jun;67(6):391–3.

129. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. New England Journal of Medicine. 2004 Sep 23;351(13):1296-305.

130. Basu G, Mohapatra A. Interactions between thyroid disorders and kidney disease. Indian J Endocrinol Metab. 2012;16(2):204.

131. Schmid C, Brändle M, Zwimpfer C, Zapf J, Wiesli P. Effect of Thyroxine Replacement on Creatinine, Insulin-Like Growth Factor 1, Acid-Labile Subunit, and Vascular Endothelial Growth Factor. Clin Chem. 2004 Jan 1;50(1):228–31.

132. Lo JC, Chertow GM, Go AS, Hsu C-Y. Increased prevalence of subclinical and clinical hypothyroidism in persons with chronic kidney disease. Kidney Int. 2005 Mar;67(3):1047–52.

133. Song SH, Kwak IS, Lee DW, Kang YH, Seong EY, Park JS. The prevalence of low triiodothyronine according to the stage of chronic kidney disease in subjects with a normal thyroid-stimulating hormone. Nephrol Dial Transplant. 2009 May 1;24(5):1534–8.

134. Wiederkehr MR, Kalogiros J, Krapf R. Correction of metabolic acidosis improves thyroid and growth hormone axes in haemodialysis patients. Nephrol Dial Transplant. 2004 May 1;19(5):1190–7.

135. Zoccali C, Tripepi G, Cutrupi S, Pizzini P, Mallamaci F. Low Triiodothyronine: A New Facet of Inflammation in End-Stage Renal Disease. J Am Soc Nephrol. 2005 Sep 1;16(9):2789–95.

136. Kaptein EM. Thyroid Hormone Metabolism and Thyroid Diseases in Chronic Renal Failure. 2018;17(1):19.

137. Hall JE, Henegar JR, Dwyer TM, Liu J, Silva AA da, Kuo JJ, et al. Is obesity a major cause of chronic kidney disease? Adv Chronic Kidney Dis. 2004 Jan 1;11(1):41–54.

138. Ejerblad E, Fored CM, Lindblad P, Fryzek J, McLaughlin JK, Nyrén O. Obesity and Risk for Chronic Renal Failure. J Am Soc Nephrol. 2006 Jun 1;17(6):1695–702.

139. Iseki K, Ikemiya Y, Kinjo K, Inoue T, Iseki C, Takishita S. Body mass index and the risk of development of end-stage renal disease in a screened cohort. Kidney Int. 2004 May 1;65(5):1870–6.

140. Hsu C, McCulloch CE, Iribarren C, Darbinian J, Go AS. Body Mass Index and Risk for End-Stage Renal Disease. Ann Intern Med. 2006 Jan 3;144(1):21.

141. Bonnet F, Deprele C, Sassolas A, Moulin P, Alamartine E, Berthezène F, et al. Excessive body weight as a new independent risk factor for clinical and pathological progression in primary IgA nephritis. Am J Kidney Dis. 2001 Apr 1;37(4):720–7.

142. Gonzalez E, Gutierrez E, Morales E, Hernandez E, Andres A, Bello I, et al. Factors influencing the progression of renal damage in patients with unilateral renal agenesis and remnant kidney. Kidney Int. 2005 Jul 1;68(1):263–70.

143. Praga M, Hernández E, Herrero JC, Morales E, Revilla Y, Díaz-González R, et al. Influence of obesity on the appearance of proteinuria and renal insufficiency after unilateral nephrectomy. Kidney Int. 2000 Nov 1;58(5):2111–8.

144. Aryee C, Owiredu WK, Osei-Yeboah J, Owusu-Dabo E, Laing EF, Owusu IK. An Analysis of Anthropometric Indicators and Modifiable Lifestyle Parameters Associated with Hypertensive Nephropathy. International journal of hypertension. 2016;2016.

145. Ram R, Goswami DN, Bhattacharya SK, Bhattacharya K, Saha BK, Saha J, et al. An epidemiological study on risk factors of diabetes mellitus among patients attending a medical college hospital in Kolkata, West Bengal. J Indian Med Assoc. 2006 Aug;104(8):428–30.

146. Srivastava T. Nondiabetic consequences of obesity on kidney. Pediatr Nephrol. 2006 Apr 1;21(4):463–70.

147. De Jong PE, Verhave JC, Pinto-Sietsma SJ, Hillege HL. Obesity and target organ damage: the kidney. International journal of obesity. 2002 Nov 28;26(S4):S21.

148. Henegar JR, Bigler SA, Henegar LK, Tyagi SC, Hall JE. Functional and Structural Changes in the Kidney in the Early Stages of Obesity. J Am Soc Nephrol. 2001 Jun 1;12(6):1211–7.

149. Wahba IM, Mak RH. Obesity and Obesity-Initiated Metabolic Syndrome: Mechanistic Links to Chronic Kidney Disease. Clin J Am Soc Nephrol. 2007 May 1;2(3):550–62.

150. Hall JE, Brands MW, Dixon WN, Smith MJ. Obesity-induced hypertension. Renal function and systemic hemodynamics. Hypertension. 1993 Sep 1;22(3):292–9.

151. Wolf G, Chen S, Han DC, Ziyadeh FN. Leptin and renal disease. Am J Kidney Dis. 2002 Jan 1;39(1):1–11.

152. Hall JE. Louis K. Dahl Memorial Lecture. Renal and cardiovascular mechanisms of hypertension in obesity. Hypertension. 1994 Mar 1;23(3):381–94.

153. Hall JE. Mechanisms of Abnormal Renal Sodium Handling in Obesity Hypertension. 2018;7.

154. Hall JE, Crook ED, Jones DW, Wofford MR, Dubbert PM. Mechanisms of Obesity-Associated Cardiovascular and Renal Disease. Am J Med Sci. 2002 Sep 1;324(3):127–37.

155. Landsberg L, Krieger DR. Obesity, Metabolism, and the Sympathetic Nervous System. Am J Hypertens. 1989 Mar 1;2(3_Pt_2):125S-132S.

156. Ferrannini E, Haffner SM, Stern MP. Essential hypertension: an insulin-resistant state. J Cardiovasc Pharmacol. 1990;15 Suppl 5:S18-25.

157. Robles RG, Villa E, Santirso R, Martinez J, Ruilope LM, Carmen C, et al. Effects of Captopril on Sympathetic Activity, Lipid and Carbohydrate Metabolism in a Model of Obesity-Induced Hypertension in Dogs. Am J Hypertens. 1993 Dec 1;6(12):1009–15.

158. Unger RH, Scherer PE. Gluttony, sloth and the metabolic syndrome: a roadmap to lipotoxicity. Trends Endocrinol Metab. 2010 Jun 1;21(6):345–52.

159. Tonelli M, Moyé L, Sacks FM, Cole T, Curhan GC, Investigators for the C and RE (CARE) T. Effect of Pravastatin on Loss of Renal Function in People with Moderate Chronic Renal Insufficiency and Cardiovascular Disease. J Am Soc Nephrol. 2003 Jun 1;14(6):1605–13.

160. Wisse BE. The Inflammatory Syndrome: The Role of Adipose Tissue Cytokines in Metabolic Disorders Linked to Obesity. J Am Soc Nephrol. 2004 Nov 1;15(11):2792–800.

161. Dandona P, Aljada A, Chaudhuri A, Mohanty P, Garg R. Metabolic Syndrome: A Comprehensive Perspective Based on Interactions Between Obesity, Diabetes, and Inflammation. Circulation. 2005 Mar 22;111(11):1448–54.

162. Wu Y, Liu Z, Xiang Z, Zeng C, Chen Z, Ma X, et al. Obesity-Related Glomerulopathy: Insights from Gene Expression Profiles of the Glomeruli Derived from Renal Biopsy Samples. Endocrinology. 2006 Jan;147(1):44–50.

163. Sharma K, Considine RV. The Ob protein (leptin) and the kidney. Kidney Int. 1998 Jun;53(6):1483–7.

164. Briley LP, Szczech LA. Leptin and Renal Disease. Semin Dial. 19(1):54–9.

165. Gal CS-L, Raufaste D, Brossard G, Pouzet B, Marty E, Maffrand J-P, et al. Characterization and localization of leptin receptors in the rat kidney. FEBS Lett. 404(2–3):185–91.

166. Wolf G, Hamann A, Han DC, Helmchen U, Thaiss F, Ziyadeh FN, et al. Leptin stimulates proliferation and TGF-β expression in renal glomerular endothelial cells: Potential role in glomerulosclerosis1. Kidney Int. 1999 Sep 1;56(3):860–72.

167. Wallace AM, McMahon AD, Packard CJ, Kelly A, Shepherd J, Gaw A, et al. Plasma Leptin and the Risk of Cardiovascular Disease in the West of Scotland Coronary Prevention Study (WOSCOPS). Circulation. 2001 Dec 18;104(25):3052–6.

168. Hamm LL, McCullough PA, Kasiske BL, Kelepouris E, Klag MJ, Parfrey P, Pfeffer M, Raij L, Spinosa DJ, Wilson PW. Kidney disease as a risk factor for development of cardiovascular disease. Circulation. 2003;108:2154-69.

169. Weiner DE, Tighiouart H, Stark PC, Amin MG, MacLeod B, Griffith JL, Salem DN, Levey AS, Sarnak MJ. Kidney disease as a risk factor for recurrent cardiovascular disease and mortality1. American Journal of Kidney Diseases. 2004 Aug 1;44(2):198-206.

170. Bleyer AJ, Shemanski LR, Burke GL, Hansen KJ, Appel RG. Tobacco, hypertension, and vascular disease: Risk factors for renal functional decline in an older population. Kidney Int. 2000 May 1;57(5):2072–9.

171. Fox CS, Larson MG, Leip EP, Culleton B, Wilson PWF, Levy D. Predictors of New-Onset Kidney Disease in a Community-Based Population. JAMA. 2004 Feb 18;291(7):844–50.

172. Brosius FC, Hostetter TH, Kelepouris E, Mitsnefes MM, Moe SM, Moore MA, et al. Detection of Chronic Kidney Disease in Patients With or at Increased Risk of Cardiovascular Disease: A Science Advisory From the American Heart Association Kidney and Cardiovascular Disease Council; the Councils on High Blood Pressure Research, Cardiovascular Disease in the Young, and Epidemiology and Prevention; and the Quality of Care and Outcomes Research Interdisciplinary Working Group: Developed in Collaboration With the National Kidney Foundation. Circulation. 2006 Sep 5;114(10):1083–7.

173. Neumann J, Ligtenberg G, Klein II, Koomans HA, Blankestijn PJ. Sympathetic hyperactivity in chronic kidney disease: Pathogenesis, clinical relevance, and treatment. Kidney Int. 2004 May 1;65(5):1568–76.

174. Xu J, Li G, Wang P, Velazquez H, Yao X, Li Y, et al. Renalase is a novel, soluble monoamine oxidase that regulates cardiac function and blood pressure. J Clin Invest. 2005 May 2;115(5):1275–80.

175. Amann K, Tenyi GMT-M, Simonoviciene A, Koch A, Orth S, Ritz E. Remodeling of Resistance Arteries in Renal Failure: Effect of Endothelin Receptor Blockade. J Am Soc Nephrol. 2001 Oct 1;12(10):2040–50.

176. Mallamaci F, Tripepi G, Maas R, Malatino L, Böger R, Zoccali C. Analysis of the Relationship between Norepinephrine and Asymmetric Dimethyl Arginine Levels among Patients with End-Stage Renal Disease. J Am Soc Nephrol. 2004 Feb 1;15(2):435–41.

177. Tonelli M, Sacks F, Pfeffer M, Gao Z, Curhan G. Relation Between Serum Phosphate Level and Cardiovascular Event Rate in People With Coronary Disease. Circulation. 2005 Oct 25;112(17):2627–33.

178. Herzog CA, Asinger RW, Berger AK, Charytan DM, Díez J, Hart RG, et al. Cardiovascular disease in chronic kidney disease. A clinical update from Kidney Disease: Improving Global Outcomes (KDIGO). Kidney Int. 2011 Sep;80(6):572–86.

179. Merkin SS, Diez Roux AV, Coresh J, Fried LF, Jackson SA, Powe NR. Individual and neighborhood socioeconomic status and progressive chronic kidney disease in an elderly population: The Cardiovascular Health Study. Soc Sci Med. 2007 Aug 1;65(4):809–21.

180. Vart P, Gansevoort RT, Coresh J, Reijneveld SA, Bültmann U. Socioeconomic Measures and CKD in the United States and The Netherlands. Clin J Am Soc Nephrol. 2013 Jun 27;CJN.12521212.

181. Nuyts GD, D'Haese PC, Elseviers MM, de Broe ME, Van Vlem E, Thys J, et al. New occupational risk factors for chronic renal failure. The Lancet. 1995 Jul 1;346(8966):7–11.

182. Stengel B, Cénée S, Limasset J-C, Protois J-C, Marcelli A, Brochard P, et al. Organic Solvent Exposure May Increase the Risk of Glomerular Nephropathies with Chronic Renal Failure. Int J Epidemiol. 1995 Apr 1;24(2):427–34.

183. Wedeen RP. Occupational and environmental renal disease. Semin Nephrol. 1997 Jan;17(1):46–53.

184. Torres C, Aragón A, González M, López I, Jakobsson K, Elinder C-G, et al. Decreased Kidney Function of Unknown Cause in Nicaragua: A Community-Based Survey. Am J Kidney Dis. 2010 Mar 1;55(3):485–96.

185. Schrier RW. Renal, Metabolic, and Circulatory Responses to Heat and Exercise: Studies in Military Recruits During Summer Training, with Implications for Acute Renal Failure. Ann Intern Med. 1970 Aug 1;73(2):213.

186. Brikowski TH, Lotan Y, Pearle MS. Climate-related increase in the prevalence of urolithiasis in the United States. Proc Natl Acad Sci. 2008 Jul 15;105(28):9841–6.

187. Wijkström J, Leiva R, Elinder C-G, Leiva S, Trujillo Z, Trujillo L, et al. Clinical and Pathological Characterization of Mesoamerican Nephropathy: A New Kidney Disease in Central America. Am J Kidney Dis. 2013 Nov 1;62(5):908–18.

188. Roncal Jimenez CA, Ishimoto T, Lanaspa MA, Rivard CJ, Nakagawa T, Ejaz AA, et al. Fructokinase activity mediates dehydration-induced renal injury. Kidney Int. 2014 Aug;86(2):294–302.

189. Cirillo P, Gersch MS, Mu W, Scherer PM, Kim KM, Gesualdo L, et al. Ketohexokinase-Dependent Metabolism of Fructose Induces Proinflammatory Mediators in Proximal Tubular Cells. J Am Soc Nephrol. 2009 Mar 1;20(3):545–53.

190. Glushakova O, Kosugi T, Roncal C, Mu W, Heinig M, Cirillo P, et al. Fructose Induces the Inflammatory Molecule ICAM-1 in Endothelial Cells. J Am Soc Nephrol. 2008 Sep 1;19(9):1712–20.

191. Wu M-S,Yang C-W, Pan M-J, Chang C-T, Chen Y-C. Reduced renal Na+-K+-Cl- co-transporter activity and inhibited NKCC2 mRNA expression by Leptospira shermani: from bed-side to bench. Nephrol Dial Transplant. 2004 Oct 1;19(10):2472–9.

192. Abdulkader RCRM, Silva MV. The kidney in leptospirosis. Pediatr Nephrol. 2008 Dec 1;23(12):2111.

193. Romieu I, Palazuelos E, Hernandez Avila M, Rios C, Muñoz I, Jimenez C, et al. Sources of lead exposure in Mexico City. Environ Health Perspect. 1994 Apr;102(4):384–9.

194. Sabolić I. Common Mechanisms in Nephropathy Induced by Toxic Metals. Nephron Physiol. 2006;104(3):p107–14.

195. Xia J, Wang L, Ma Z, Zhong L, Wang Y, Gao Y, et al. Cigarette smoking and chronic kidney disease in the general population: a systematic review and meta-analysis of prospective cohort studies. Nephrol Dial Transplant. 2017 Mar 1;32(3):475–87.

196. Orth SR. Cigarette smoking: an important renal risk factor–far beyond carcinogenesis. Tobacco induced diseases. 2003 Jun;1(2):137.

197. Orth SR. Effects of Smoking on Systemic and Intrarenal Hemodynamics: Influence on Renal Function. J Am Soc Nephrol. 2004 Jan 1;15(1 suppl):S58–63.

198. Orth SR. Smoking and the Kidney. J Am Soc Nephrol. 2002 Jun 1;13(6):1663–72.

199. Baggio B, Budakovic A, Perissinotto E, Maggi S, Cantaro S, Enzi G, et al. Atherosclerotic risk factors and renal function in the elderly: The role of hyperfibrinogenaemia and smoking. Results from the Italian Longitudinal Study on Ageing (ILSA). Nephrol Dial Transplant Off Publ Eur Dial Transpl Assoc - Eur Ren Assoc. 2005 Feb 1;20:114–23.

200. Jaimes EA, Tian R-X, Raij L. Nicotine: the link between cigarette smoking and the progression of renal injury? Am J Physiol Heart Circ Physiol. 2007 Jan;292(1):H76-82.

201. Dasgupta P, Chellappan SP. Nicotine-mediated cell proliferation and angiogenesis: new twists to an old story. Cell Cycle Georget Tex. 2006 Oct;5(20):2324–8.

202. Grassi G, Seravalle G, Calhoun DA, Bolla GB, Giannattasio C, Marabini M, et al. Mechanisms responsible for sympathetic activation by cigarette smoking in humans. Circulation. 1994 Jul 1;90(1):248–53.

203. Hansen KW, Pedersen MM, Christiansen JS, Mogensen CE. Night Blood Pressure and Cigarette Smoking: Disparate Association in Healthy Subjects and Diabetic Patients. Blood Press. 1994 Jan 1;3(6):381–8.

204. Davidson MB, Hix JK, Vidt DG, Brotman DJ. Association of Impaired Diurnal Blood Pressure Variation With a Subsequent Decline in Glomerular Filtration Rate. Arch Intern Med. 2006 Apr 24;166(8):846–52.

205. Ritz E, Benck U, Franek E, Keller C, Seyfarth M, Clorius J. Effects of smoking on renal hemodynamics in healthy volunteers and in patients with glomerular disease. J Am Soc Nephrol. 1998 Oct 1;9(10):1798–804.

206. Main J. How Important Is Atheromatous Renal Artery Stenosis as a Cause of End-Stage Renal Disease? Semin Dial. 14(3):143–5.

207. Auerbach O, Carter HW, Garfinkel L, Hammond EC. Cigarette Smoking and Coronary Artery Disease: A Macroscopic and Microscopic Study. Chest. 1976 Dec 1;70(6):697–705.

208. Bos WJW, Demircan MM, Weening JJ, Krediet RT, Wal VD, C A. Renal vascular changes in renal disease independent of hypertension. Nephrol Dial Transplant. 2001 Mar 1;16(3):537–41.

209. Markowitz GS, Lin J, Valeri AM, Avila C, Nasr SH, D'Agati VD. Idiopathic nodular glomerulosclerosis is a distinct clinicopathologic entity linked to hypertension and smoking. Hum Pathol. 2002 Aug 1;33(8):826–35.

210. World Health Organization. Substance Abuse Dept, World Health Organization. Department of Mental Health, Substance Abuse, World Health Organization. Global status report: alcohol policy. World Health Organization; 2004 Jul 23.

211. Thiel DHV, Gavaler JS, Little JM, Lester R. Alcohol: Its effect on the kidney. Metab - Clin Exp. 1977 Aug 1;26(8):857–66.

212. Keller CK, Andrassy K, Waldherr R, Ritz E. Postinfectious glomerulonephritis—is there a link to alcoholism? QJM Int J Med. 1994 Feb 1;87(2):97–102.

213. Pablo NC, Churg J, Needle MA, Ganesharajah M. Renal Papillary Necrosis: Relapsing Form Associated With Alcoholism. Am J Kidney Dis. 1986 Jan 1;7(1):88–94.

214. Muthukumar T, Jha V, Wanchoo A, Bambery P, Sakhuja V. Acute Renal Failure Due to Nontraumatic Rhabdomyolysis Following Binge Drinking. Ren Fail. 1999 Jan;21(5):545–9.

215. Cheungpasitporn W, Thongprayoon C, Kittanamongkolchai W, Brabec BA, O'Corragain OA, Edmonds PJ, et al. High alcohol consumption and the risk of renal damage: a systematic review and meta-analysis. QJM Int J Med. 2015 Jul 1;108(7):539–48.

216. Tanasescu M, Hu FB, Willett WC, Stampfer MJ, Rimm EB. Alcohol consumption and risk of coronary heart disease among men with type 2 diabetes mellitus. J Am Coll Cardiol. 2001 Dec 1;38(7):1836–42.

217. Gaziano JM, Buring JE, Breslow JL, Goldhaber SZ, Rosner B, Van Denburgh M, et al. Moderate Alcohol Intake, Increased Levels of High-Density Lipoprotein and Its Subfractions, and Decreased Risk of Myocardial Infarction. J Occup Environ Med. 1994 May;36(5):504.

218. Rodrigo R, Rivera G, Orellana M, Araya J, Bosco C. Rat kidney antioxidant response to long-term exposure to flavonol rich red wine. Life Sci. 2002 Nov 1;71(24):2881–95.

219. Shigematsu S, Ishida S, Hara M, Takahashi N, Yoshimatsu H, Sakata T, et al. Resveratrol, a red wine constituent polyphenol, prevents superoxide-dependent inflammatory responses induced by ischemia/reperfusion, platelet-activating factor, or oxidants. Free Radic Biol Med. 2003 Apr 1;34(7):810–7.

220. Hippisley-Cox J, Coupland C. Predicting the risk of Chronic Kidney Disease in Men and Women in England and Wales: prospective derivation and external validation of the QKidney® Scores. BMC family practice. 2010 Dec;11(1):49.

221. Rule AD, Bergstralh EJ, Melton LJ, Li X, Weaver AL, Lieske JC. Kidney Stones and the Risk for Chronic Kidney Disease. Clin J Am Soc Nephrol. 2009 Apr 1;4(4):804–11.

222. Jungers P, Joly D, Barbey F, Choukroun G, Daudon M. ESRD caused by nephrolithiasis: Prevalence, mechanisms, and prevention. Am J Kidney Dis. 2004 Nov 1;44(5):799–805.

223. Saucier NA, Sinha MK, Liang KV, Krambeck AE, Weaver AL, Bergstralh EJ, et al. Risk Factors for CKD in Persons With Kidney Stones: A Case-Control Study in Olmsted County, Minnesota. Am J Kidney Dis. 2010 Jan 1;55(1):61–8.

224. Evan AP, Lingeman JE, Coe FL, Shao Y, Parks JH, Bledsoe SB, et al. Crystal-associated nephropathy in patients with brushite nephrolithiasis. Kidney Int. 2005 Feb;67(2):576–91.

225. Klahr S. New Insights Into the Consequences and Mechanisms of Renal Impairment in Obstructive Nephropathy. Am J Kidney Dis. 1991 Dec;18(6):689–99.

226. Hwang SJ, Haas M, Harris HW, Silva P, Yalla S, Sullivan MR, Otuechere G, Kashgarian M, Zeidel ML. Transport defects of rabbit medullary thick ascending limb cells in obstructive nephropathy. The Journal of clinical investigation. 1993 Jan 1;91(1):21-8.

227. Ophascharoensuk V, Giachelli CM, Gordon K, Hughes J, Pichler R, Brown P, et al. Obstructive uropathy in the mouse: Role of osteopontin in interstitial fibrosis and apoptosis. Kidney Int. 1999 Aug;56(2):571–80.

228. Larsson PA, Cano M, Grenabo L, Brorson J-E, Hedelin H, Pettersson S, et al. Morphological Lesions of the Rat Urinary Tract Induced by Inoculation of Mycoplasmas and Other Urinary Tract Pathogens. Urol Int. 1989;44(4):210–7.

229. Johnson RJ, Kivlighn SD, Kim Y-G, Suga S, Fogo AB. Reappraisal of the pathogenesis and consequences of hyperuricemia in hypertension, cardiovascular disease, and renal disease. Am J Kidney Dis. 1999 Feb 1;33(2):225–34.

230. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL, et al. Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. 2003 Dec 1;42(6):1206–52.

231. Lim JU, Lee JH, Kim JS, Hwang YI, Kim T-H, Lim SY, et al. Comparison of World Health Organization and Asia-Pacific body mass index classifications in COPD patients. Int J Chron Obstruct Pulmon Dis. 2017 Aug 21;12:2465–75.

232. American Diabetes Association. 2. Classification and Diagnosis of Diabetes. Diabetes Care. 2017 Jan;40(Supplement 1):S11–24.

233. Singh NP, Ingle GK, Saini VK, Jami A, Beniwal P, Lal M, Meena GS. Prevalence of low glomerular filtration rate, proteinuria and associated risk factors in North India using Cockcroft-Gault and Modification of Diet in Renal Disease equation: an observational, cross-sectional study. BMC nephrology. 2009 Dec;10(1):4

234. Varma PP, Raman DK, Ramakrishnan TS, Singh P, Varma A. Prevalence of early stages of chronic kidney disease in apparently healthy central government employees in India. Nephrol Dial Transplant. 2010 Sep;25(9):3011–7.

235. 235. Agarwal SK, Dash SC, Irshad M, Raju S, Singh R, Pandey RM. Prevalence of chronic renal failure in adults in Delhi, India. Nephrol Dial Transplant. 2005 Aug 1;20(8):1638–42.

236. Vassalotti JA, Li S, Chen S-C, Collins AJ. Screening Populations at Increased Risk of CKD: The Kidney Early Evaluation Program (KEEP) and the Public Health Problem. Am J Kidney Dis. 2009 Mar 1;53(3, Supplement 3):S107–14.

237. Inaguma D, Imai E, Takeuchi A, Ohashi Y, Watanabe T, et al. Risk factors for CKD progression in Japanese patients: findings from the Chronic Kidney Disease Japan Cohort (CKD-JAC) study. Clin Exp Nephrol. 2017 Jun;21(3):446–56.

238. Ying T, Clayton P, Naresh C, Chadban S. Predictive value of spot versus 24-hour measures of proteinuria for death, end-stage kidney disease or chronic kidney disease progression. BMC nephrology. 2018 Dec;19(1):55.

239. Chang YC, Chang CH, Yeh YC, Chuang LM, Tu YK. Subclinical and overt hypothyroidism is associated with reduced glomerular filtration rate and proteinuria: a large cross-sectional population study. Scientific reports. 2018 Feb 1;8(1):2031.

240. Khatiwada S, Rajendra KC, Gautam S, Lamsal M, Baral N. Thyroid dysfunction and dyslipidemia in chronic kidney disease patients. BMC Endocrine disorders. 2015 Dec;15(1):65

241. Vargas F, Moreno JM, Rodríguez-Gómez I, Wangensteen R, Osuna A, Álvarez-Guerra M, et al. Vascular and renal function in experimental thyroid disorders. Eur J Endocrinol. 2006 Feb 1;154(2):197–212.

242. Zaman SB, Hossain N, Rahman M. Associations between Body Mass Index and Chronic Kidney Disease in Type 2 Diabetes Mellitus Patients: Findings from the Northeast of Thailand. :9.

243. Jaroszynski A, Dereziński T, Jaroszyńska A, Zapolski T, Wąsikowska B, Wysokiński A, et al. Association of anthropometric measures of obesity and chronic kidney disease in elderly women. Ann Agric Environ Med. 2016 Sep 27;23(4):636–40.

244. Wu B, Bell K, Stanford A, Kern DM, Tunceli O, Vupputuri S, et al. Understanding CKD among patients with T2DM: prevalence, temporal trends, and treatment patterns—NHANES 2007–2012. BMJ Open Diabetes Res Care. 2016 Apr;4(1):e000154.

245. Gatwood J, Chisholm-Burns M, Davis R, Thomas F, Potukuchi P, Hung A, et al. Evidence of chronic kidney disease in veterans with incident diabetes mellitus. Reboldi G, editor. PLOS ONE. 2018 Feb 9;13(2):e0192712.

246. Sharma M, Doley P, Das HJ. Etiological profile of chronic kidney disease: A single-center retrospective hospital-based study. Saudi J Kidney Dis Transplant. 2018 Mar 1;29(2):409.

247. Soman SK, Areekal B, Varghese RG, John S, Gopan RS, Jacob QM, et al. Chronic kidney disease-prevalence and determinants among type 2 diabetes mellitus patients attending a primary care setting in central Kerala. Int J Community Med Public Health. 2018 Jun 22;5(7):2776–81.

248. Ruilope LM, Salvetti A, Jamerson K, Hansson L, Warnold I, Wedel H, et al. Renal Function and Intensive Lowering of Blood Pressure in Hypertensive Participants of the Hypertension Optimal Treatment (HOT) Study. J Am Soc Nephrol. 2001 Feb 1;12(2):218–25.

249. Mann JFE, Gerstein HC, Pogue J, Bosch J, Yusuf S, for the HOPE Investigators. Renal Insufficiency as a Predictor of Cardiovascular Outcomes and the Impact of Ramipril: The HOPE Randomized Trial. Ann Intern Med. 2001 Apr 17;134(8):629.

250. Culleton BF, Larson MG, Wilson PWF, Evans JC, Parfrey PS, Levy D. Cardiovascular disease and mortality in a community-based cohort with mild renal insufficiency. Kidney Int. 1999 Dec;56(6):2214–9.

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
1	55	M	1.53	51	80	21.79	140	90	68	Y	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	68	110		5.9	26	1.1	186	220	40	143	37	0.99	7.77	3.3	0.16	75.2
2	75	F	1.5	60	96	26.67	120	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	250	294		8.1	41	0.9	73	114	35	65	15	2.56	1.44	2.78	0.52	62.5
3	57	F	1.52	55	91	23.81	160	100	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	144	177		7.3	26	0.9	192	161	44	89	38	2.03	1.24	3.15	0.76	71
4	65	F	1.5	49	78	21.78	120	60	68	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			100		26	1	102	219	37	144	20	1.07	7.77	1.97	1.81	59.1
5	50	M	1.59	51	79	20.17	140	80	72	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	196	348		8.8	33	1.2	286	219	40	130	57	2.75	5.56	3.4	0.1	70.1
6	35	M	1.6	70.5	92	27.54	120	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	81	112			16	1.2	124	184	33	121	25	1.79	1.46	3.94	0.01	76.8
7	68	F	1.49	53	87	23.87	140	70	78	N	N	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	157	225		6.8	19	0.9	129	87	24	46	26	0.15	2.06	3.78	0.11	65.7
8	64	M	1.6	56	86	21.88	100	60	80	y	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	52	85	220	8.2	57	2.3	96	113	22	73	19	0.71	1.46	2.47	3.79	28.9
9	35	M	1.84	88	98	25.99	120	80	82	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	86	142			28	1.7	81	140	33	82	16	3.5	1.22	3.97	0.09	51.1
10	56	M	1.71	66.8	93	22.84	130	90	88	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N	116	144			19	1.2	132	129	25	76	26	3.57	1.26	3.45	0.04	67.2
11	55	M	1.76	63.4	86	20.47	130	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	74	193			31	1.1	57	121	35	69	11	4.02	1.26	3.27	0.05	75.2
12	49	F	1.56	78.8	113	32.38	110	80	86	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	109	194			27	1.09	152	176	42	101	30	0.89	1.18	2.98	0.04	59.6
13	52	M	1.63	75	100	28.23	120	80	78	N	N	N	N	N	N	N	N	N	N	N	y	N	N	N	N	113	243		6.6	18	0.9	218	175	33	101	44	2.75	1.26	2.8	0.07	97.9
14	47	M	1.58	68	92	27.24	130	80	86	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Y	Y	N			113		45	2.7	128	143	39	77	26	5.02	3.81	2.26	4.5	26.9
15	43	M	1.62	61	84	23.24	120	70	84	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	92	111			26	1.08	77	147	42	84	15	1.69	1.18	3.42	0.01	83.6
16	35	F	1.56	77.5	84	31.85	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	80	102			22	0.95	54	153	47	84	11	1.69	1.3	3.08	0.01	77.6
17	39	M	1.62	59.3	74	22.60	90	60	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	78	112			27	1.13	107	163	43	94	21	2.75	1.26	2.8	0.01	81.4
18	44	F	1.49	59.2	82	26.67	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	76	119			13	0.96	47	89	25	53	9	18.88	0.75	3.02	0.01	71.9
19	59	M	1.6	53	90	20.70	110	70	86	Y	N	N	N	N	N	N	N	N	y	N	N	Y	N	Y	N			127		41	0.8	54	45	56	26	11	1.03	1.48	2.5	0.03	97.8
20	22	M	1.68	74	90	26.22	110	70	74	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			79		15	1.2	53	125	46	64	11	0.01	1.25	2.61	0.02	85.3
21	53	M	1.65	63	95	23.14	130	90	86	N	Y	N	N	Y	N	N	N	N	N	N	y	N	Y	N	N	212	295	179	9.8	31	1.1	81	131	30	88	16	0.71	1.96	2.47	0.92	16.2
22	43	F	1.52	53	86	22.94	120	60	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	73	88			15	1.05	56	157	48	90	11	2.73	1.19	2.87	0.07	65
23	45	F	1.56	51	86	20.96	110	70	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	120	80			33	1.2	115	175	40	108	23	5.77	1.08	3.24	0.18	54.5
24	60	F	1.64	77	110	28.63	120	80	76	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	125	184			18	1.2	86	184	38	119	17	2.36	1.3	2.71	0.08	59.7
25	50	M	1.72	77	98	26.03	120	70	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	101	79			18	1.26	495	201	39	101	99	1.21	1.24	3.36	0.06	66.1
26	60	M	1.69	77	97	26.96	130	80	84	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	80	135			26	1.3	147	198	46	118	29	4.1	1.04	2.65	0.09	59.3
27	65	M	1.64	61	70	22.68	130	60	84	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	99	121			16	1.29	80	161	36	102	16	4.17	1.27	3.13	0.02	57.8
28	52	M	1.65	70	86	25.71	140	90	66	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	170			9.3	16	0.9	156	191	49	106	31	1.46	1.17	2.02	0.65	97.9
29	60	F	1.52	58	106	25.10	130	80	72	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	84	254		6.5	18	0.8	114	82	20	44	23	2.58	1.24	2.3	1.1	80.1
30	60	M	1.57	74	113	30.02	140	70	70	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	75	96			42	1.5	89	117	28	74	18	3.3	0.96	4.46	0.6	49.9
31	33	F	1.54	55	84	23.19	110	70	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			98		20	0.4	80	158	42	103	14	2.53	1.22	3.23	1.04	136.9
32	40	F	1.48	51	88	23.28	120	80	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			146		15	1	49	111	31	64	16	4.79	1.42	2.89	0.1	70.4
33	75	M	1.7	54	83	18.69	130	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	124	175	232	7.6	22	1.4	150	192	43	116	30	3.75	1.31	2.05	1.73	48.8
34	48	M	1.73	68	93	22.72	120	70	74	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	235	425		7.6	22	1.2	162	224	36	78	27	1.86	1.25	2.32	0.08	71.1
35	40	M	1.61	50	73	19.29	120	80	78	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			85		32	1.5	165	184	38	105	33	2.89	1.17	2.58	0.8	57.4
36	52	M	1.72	51	78	17.24	120	70	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	n			95		21	1.2	78	155	42	94	16	2.9	1.45	3.16	1.12	69.1
37	39	M	1.53	66	90	28.19	110	70	82	N	N	Y	N	N	Y	N	N	N	Y	Y	N	N	Y	Y	N	331	420		9.2	29	1.4	128	97	32	50	26	0.04	1.86	3.66	0.09	62.8
38	51	M	1.69	75	76	26.26	110	80	84	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N	Y	97	214		6.2	23	1.5	436	195	31	100	87	1.41	1.15	3.43	0.05	50.9
39	53	F	1.5	79.7	85	35.42	140	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	109			24	1.1	94	147	28	93	19	1.28	1.08	3.06	0.1	57.3
40	52	M	1.71	67	80	22.91	100	70	88	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	Y	N	170	314		8.2	26	1.43	88	127	33	75	18	2.57	1.25	3.05	0.09	55.9
41	58	M	1.63	65	80	24.46	130	80	84	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	91	142			19	1.4	88	147	29	94	18	1.11	1.02	2.36	0.43	55
42	52	M	1.72	68	76	22.99	130	80	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	90	116			28	1.13	72	196	45	122	14	1.08	1.18	3.37	0.06	74.3
43	50	F	1.45	56	103	26.63	110	70	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	324	414		8.6	17	0.92	113	185	39	120	23	2.39	1.29	3.19	0.93	72.6
44	52	M	1.54	78	107	32.89	110	70	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	101	153			21	1.38	95	139	38	79	19	4.09	1.25	3.34	0.07	58.4
45	31	F	1.44	62	62	29.90	120	80	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			98		20	1	113	205	41	122	23	47.04	0.71	2.34	0.09	75
46	24	M	1.63	44	63	16.56	110	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			112	2.83	15	1.1	116	128	21	81	23	1.04	1.69	2.22	0.12	93.5
47	20	M	1.495	41.5	73	18.57	130	80	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			74		16	1	69	171	37	93	14	2.33	1.86	3.66	0.12	107.9
48	26	F	1.46	65	102	30.49	110	70	74	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			76		18	0.92	68	182	42	108	42	4.78	1.1	2.74	0.09	85.9
49	32	F	1.5	57	83	25.33	110	80	78	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N			86		24	0.9	84	118	37	64	17	47.64	0.71	2.34	0.05	84.6
50	56	F	1.44	49	94	23.63	180	110	80	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			84		16	1	85	181	43	101	17	1.58	1.48	2.88	0.23	62.9
51	46	M	1.56	59	88	24.24	110	70	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	107	133			28	1.16	103	188	41	114	21	2.67	1.09	2.64	0.14	75.1

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
52	29	F	1.5	68.6	92	30.49	110	80	84	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	98	113			18	0.88	43	121	38	72	9	1	1.55	2.89	0.08	90
53	26	F	1.57	92.5	124	37.53	110	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	86	124			19	0.78	102	133	35	79	20	9.41	1.07	3.21	0.08	104.9
54	61	M	1.56	63	93	25.89	170	80	84	N	Y	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	88	140			37	1.3	97	138	44	75	19	2.12	1.31	3.06	0.06	58.9
55	61	M	1.66	62	91	22.50	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	93	109			25	1.18	77	169	40	104	15	1.69	1.4	3.23	0.14	66.2
56	60	M	1.61	54.6	78	21.06	140	100	84	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	99	131			34	2.04	98	138	42	79	20	0.9	1.75	3.28	0.3	34.4
57	41	M	1.65	73.7	90	27.07	130	90	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	95	85			25	1.4	98	167	50	92	20	2.36	1.25	2.89	0.04	62
58	27	M	1.66	49.3	67	17.89	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	95	132			17	1.09	49	161	60	84	10	1.46	1.33	3.41	0.07	92.5
59	62	M	1.68	65	88	23.03	110	60	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	94	143			30	1.2	65	160	39	97	13	0.6	1.18	2.57	0.08	64.4
60	57	F	1.61	71.5	90	27.58	100	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	115	239		7.2	20	0.9	122	197	45	113	24	2.48	1.13	3.13	0.12	71
61	49	F	1.61	61.5	100	23.73	120	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	359	439		8.8	16	0.96	65	141	44	75	32	2.61	1.28	4.29	0.09	69.4
62	69	M	1.72	74	96	25.01	130	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	97	154			33	1.2	134	159	29	96	27	2.33	1.07	4.29	0.01	61.3
63	62	F	1.45	61.8	92	29.39	140	90	80	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	83	124			19	0.8	151	189	35	115	30	3.36	1.37	3.27	0.12	79
64	28	M	1.66	78.7	95	28.56	110	70	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	92	115			15	1.2	100	193	64	100	20	2.42	1.47	3.95	0.09	81.8
65	37	M	1.72	80.6	80	27.24	110	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	392	588		10.1	39	1.5	306	237	42	137	61	1.49	1.39	2.81	0.1	58.6
66	61	M	1.72	80.6	96	27.24	110	80	84	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	87	102			25	1.4	90	170	32	103	18	5.86	1.22	3.2	0.01	53.8
67	75	F	1.58	58	100	23.23	190	100	80	N	N	Y	Y	Y	N	Y	N	N	N	N	Y	N	N	N	Y	153	249		7.7	20	1	240	205	33	116	48	2.52	1.32	1.42	0.57	62.1
68	22	F	1.54	57	85	24.03	110	80	90	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			90		16	0.9	98	181	46	76	20	1.33	1.54	3.78	0.09	90.8
69	43	F	1.48	49	84	22.37	110	70	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			95		25	0.9	81	198	41	121	16	3.17	0.98	3.77	0.08	78.3
70	20	F	1.42	31	60	15.37	100	70	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			101		25	0.9	79	115	31	63	16	3	1.27	3.58	0.12	92
71	70	F	1.51	49	86	21.49	110	70	72	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	139	305	>500	12.8	30	1.6	87	115	37	61	17	3.9	1.03	2.31	0.003	32.3
72	70	M	1.72	80	98	27.04	120	80	84	N	Y	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	103	138			20	1.3	217	214	36	128	43	4.51	1.27	2.69	0.08	55.3
73	40	F	1.55	80	109	33.30	130	90	86	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	88	142			15	0.9	114	157	43	85	23	4.32	1.17	2.91	0.07	80
74	52	M	1.57	66	98	26.78	130	70	86	N	Y	Y	N	N	N	N	Y	N	N	N	Y	N	N	Y	N	98	172		6.9	15	1	105	109	29	57	21	6.55	1.59	2.03	0.26	86.2
75	40	M	1.69	57.2	78	20.03	110	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	84	129			16	1.1	186	210	49	121	37	0.82	1.31	2.81	0.14	83.5
76	55	M	1.65	119	134	43.71	150	80	80	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	211	271		8.1	27	1.9	120	239	54	144	24	1.36	1.53	1.69	0.3	29.2
77	65	F	1.43	41	80	20.05	130	100	86	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N			79		22	1	73	154	55	81	15	1.34	1.58	2.14	0.17	59.1
78	55	M	1.68	86	96	30.47	110	80	74	N	N	Y	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N	161	215		6.7	22	1.1	180	189	29	116	36	6.79	1.23	3.04	0.08	75.2
79	45	F	1.53	51	94	21.79	120	70	80	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N			77		16	1	116	209	55	118	23	2.9	1	3.71	0.07	68
80	58	M	1.66	84	111	30.48	140	90	84	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	115	95			15	1.04	86	177	46	99	17	2.48	1.27	2.17	0.08	78.8
81	37	F	1.65	78	102	28.65	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	109	128			16	0.98	72	139	40	78	14	2.34	1.04	2.62	0.04	73.7
82	36	M	1.725	57.8	81	19.42	100	60	80	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	90	116			16	1	60	141	33	87	12	1.22	1.21	3.14	0.1	96.4
83	25	M	1.65	70	78	25.71	130	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	89	99			16	1.22	116	181	43	109	23	3.51	1.19	2.13	0.05	81.9
84	55	F	1.47	47	82	21.75	100	60	78	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	90	129			30	1.2	253	201	43	110	51	100	0.13	0.29	0.07	50.8
85	33	F	1.63	48	76	18.07	100	80	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	119			18	0.87	73	162	46	91	15	1.8	1.2	2.9	0.06	87.5
86	40	F	1.55	72	106	29.97	120	80	76	N	N	N	N	N	N	N	N	N	N	N	y	N	N	N	Y	82	106			18	1.01	85	158	39	92	17	12.87	0.8	2.85	0.12	69.1
87	35	F	1.5	52.5	85	23.33	110	80	80	N	N	N	N	N	N	N	N	N	N	N	y	N	N	N	N	91	85			19	0.97	106	173	43	105	21	3.48	1.16	3.23	0.1	75.7
88	38	F	1.48	50	86	22.83	110	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			116		15	0.97	106	173	43	105	21	1.91	1.61	3.25	0.07	74.1
89	50	F	1.55	78	78	32.47	130	90	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	86	127			21	1	88	172	38	108	18	2.93	1.07	3.39	0.1	65.6
90	48	F	1.44	48	101	23.15	110	80	66	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	132	170		8.8	18	1.2	137	260	53	162	27	0.8	1.51	2.24	0.13	53.4
91	53	F	1.55	68	86	28.30	130	90	80	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	93	153			25	1.14	206	204	40	118	41	3.61	1.42	2.95	0.18	54.9
92	48	F	1.53	65	94	27.77	120	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	162	172		8.2	15	0.9	131	174	47	100	26	1.96	1.36	2.61	0.04	75.6
93	54	M	1.65	93	113	34.16	160	100	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	91	113			19	1.23	101	175	43	103	20	1.87	1.05	2.97	0.06	66.1
94	45	F	1.55	63	93	26.22	130	80	96	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	124	152		6.48	16	0.96	135	165	27	105	27	5.6	1.36	3.26	0.1	71.4
95	27	M	1.75	72.5	75	23.67	110	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	93	105			18	1.13	233	212	38	124	47	1.22	1.21	3.14	0.06	88.6
96	20	F	1.5	56	86	24.89	90	60	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			74		15	0.89	46	105	34	61	9	2.75	1.09	3.42	0.25	93.3
97	40	M	1.6	68	94	26.56	130	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	91	124		4.95	22	1.01	92	148	39	87	18	3.3	1.07	3.71	0.16	92.6
98	63	M	1.6	65	101	25.39	130	80	86	N	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	101	169		5.71	39	1.09	273	123	22	63	55	2.03	1.09	2.81	0.1	54
99	41	M	1.6	54	80	21.09	180	80	66	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	117	129			15	1.17	126	101	36	54	25	3.57	0.99	3.74	0.19	77
100	59	F	1.6	66	87	25.78	140	80	86	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	116	156		7.1	15	1.06	244	253	47	151	49	2.62	1.31	2.62	0.1	57.4
101	47	F	1.53	74	105	31.61	120	80	66	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N			96		22	0.1	88	190	58	108	18	4.68	1.33	2.81	0.06	195.7
102	45	M	1.7	68	83	23.53	120	80	86	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	95	77			21	1.23	67	172	51	100	13	1.26	1.35	2.97	0.07	70.5

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
103	32	F	1.58	32	64	12.82	90	60	82	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N			119		16	0.7	94	61	17	32	19	1.06	0.98	2	0.53	114.6
104	39	M	1.79	70	89	21.85	120	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	250	366	460	8.6	15	1.2	267	177	35	106	53	1.58	1.38	3.09	0.08	75.7
105	60	M	1.62	68	90	25.91	150	90	82	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	134	198		8.6	23	1.3	69	160	34	99	14	1.9	1.4	2.2	0.11	59.3
106	43	M	1.64	76	94	28.26	110	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	94	89			20	1.24	106	117	25	74	21	0.83	1.49	2.88	0.1	70.8
107	65	M	1.68	62.3	94	22.07	130	80	72	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	96	181		6.8	68	2.4	82	137	41	78	16	3.31	1.27	2.77	0.1	27.3
108	37	M	1.75	75	91	24.49	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	97	140			29	1.29	161	214	46	135	32	5.26	1.09	3.42	0.07	70.4
109	69	F	1.53	68	115	29.05	150	80	68	N	Y	Y	N	Y	N	N	N	N	N	N	y	N	N	N	N	181	270		8.5	18	1.04	157	211	37	138	31	2.64	1.14	2.58	0.12	54.8
110	20	F	1.5	65	100	28.89	100	80	68	N	N	N	N	N	N	N	N	N	N	N	y	N	N	n	N			84		14	0.98	130	129	28	80	26	1.4	1.12	2.77	0.09	83
111	65	F	1.47	30	75	13.88	110	80	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			252	6.5	15	0.97	82	114	36	62	16	2.64	1.29	2.56	0.26	61.3
112	55	F	1.42	58	88	28.76	110	80	88	N	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N			149	8.5	31	1.7	172	121	24	73	34	9.55	1.52	3.06	0.15	33.4
113	70	F	1.37	38	89	20.25	160	80	74	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			112	6.6	17	0.9	64	74	39	33	13	2.61	0.92	3.48	0.39	64.8
114	47	F	1.51	55	95	24.12	130	80	70	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			87		20	0.9	174	198	55	114	35	2.24	1.16	3.06	0.07	76.1
115	36	F	1.44	40	88	19.29	120	60	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			103		15	1	65	125	38	74	13	1.28	1.23	2.8	0.19	72.4
116	49	F	1.45	39	76	18.55	90	60	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			168	5.9	21	0.9	80	193	44	123	16	4.16	1.14	2.43	0.14	75.1
117	30	M	1.68	60	78	21.26	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	90	100			17	1.34	47	126	36	77	9	2.05	1.48	3.37	0.09	70.6
118	40	F	1.58	40	76	16.02	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	77	101			18	1.17	70	149	33	94	14	3.06	1.02	3.06	0.05	58.2
119	50	M	1.64	70	88	26.03	120	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	125	341		8.5	18	1.3	238	177	47	97	48	1.54	1.49	3.6	0.05	63.6
120	37	F	1.55	40	70	16.65	120	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	83	137			15	1.01	139	148	30	90	28	5.07	1.16	3.02	0.15	71.1
121	23	F	1.45	55	92	26.16	90	60	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			98		23	0.8	46	140	49	78	9	4.4	1.32	3.17	0.03	103.9
122	50	M	1.66	74	86	26.85	120	80	64	N	N	Y	N	Y	N	N	N	N	N	N	Y	N	N	Y	N	102	211		8.5	42	2.85	118	137	30	81	24	3.04	1.07	1.25	0.35	24.6
123	43	M	1.7	65	85	22.49	110	80	86	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	180	375		8.69	21	0.9	114	228	52	139	23	2.05	1.02	1.53	0.09	104.2
124	57	F	1.5	65	98	28.89	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	101	88			17	0.83	268	224	47	128	54	2.93	0.77	5.09	0.16	78.3
125	62	M	1.54	58	76	24.46	150	90	88	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	163	202		9.11	36	1.67	127	168	40	101	25	2.52	1.12	1.78	3.47	43.2
126	26	F	1.51	46	87	20.17	100	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			102		15	0.7	54	135	45	79	11	3.18	1.02	2.92	0.32	119.6
127	50	F	1.55	64	114	26.64	140	80	82	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			104	5.4	26	2.2	167	250	59	144	33	12.34	1.47	2.48	0.19	25.3
128	35	F	1.53	52	92	22.21	110	80	70	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			151	4.9	16	0.6	134	339	67	200	27	16.75	1.04	2.13	6.9	118.1
129	68	M	1.72	76	104	25.69	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			136	6.63	25	1.33	123	211	33	137	25	6.63	0.88	3.2	0.11	54.5
130	40	F	1.52	60	102	25.97	120	70	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			99	6.75	29	1.1	83	190	41	122	17	3.39	1.07	3.05	0.12	62.8
131	58	F	1.6	70	106	27.34	110	70	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			78	6.08	20	0.9	295	202	41	114	59	7.91	1.04	2.33	0.08	70.5
132	34	M	1.7	76	102	26.30	130	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	90	113			15	1.31	156	133	37	95	31	17.63	1.25	3.15	0.05	70.4
133	42	M	1.6	64	88	25.00	130	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	90	106			22	0.99	197	156	27	95	39	2.51	1.25	1.07	0.05	93.6
134	52	M	1.72	76	95	25.69	120	80	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			92		45	1.68	77	185	37	114	15	0.33	1.19	2.86	0.14	46
135	60	M	1.58	56	74	22.43	120	80	80	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	y	N			97		45	2.7	272	215	44	109	54	1.52	1.49	3.02	0.15	24.5
136	50	M	1.6	70	92	27.34	120	80	76	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	230	364		7.3	15	0.9	110	142	34	85	22	2.97	0.93	2.91	0.14	99.2
137	63	M	1.62	83	107	31.63	140	90	72	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	y	N	104	170		7.25	36	1	71	126	38	71	14	4.25	1.3	2.27	0.03	79.7
138	46	M	1.65	65	96	23.88	110	80	78	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	Y	N	193	289		7.9	22	1.01	146	148	31	87	29	2.23	0.98	3.36	0.26	88.8
139	42	M	1.6	67	86	26.17	110	80	88	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	108	202		8.2	41	2.44	110	149	40	86	22	1.53	1.55	2.95	1.77	31.4
140	43	M	1.45	54	90	25.68	110	70	76	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	103	114			15	0.84	111	122	24	72	22	6.91	0.83	2.75	0.11	85.1
141	56	M	1.52	60	93	25.97	110	60	88	N	Y	Y	N	N	N	N	N	N	N	N	y	N	N	N	N			186	10.6	28	1.21	177	198	32	98	25	4.34	1.02	2.62	0.16	66.5
142	52	F	1.63	99.6	120	37.49	130	90	86	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N			166	8.2	15	1	196	250	38	101	42	4.67	1.24	2.9	0.23	64.7
143	39	F	1.56	63	99	25.89	100	70	99	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			108		15	0.8	135	94	13	58	27	3.12	1.11	2.43	0.19	92.9
144	50	F	1.68	74	112	26.22	140	100	78	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	90	99	129		15	0.9	71	185	39	117	14	2.59	1.32	1.49	0.13	74.6
145	46	M	1.68	76	88	26.93	120	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	135	159		7.6	29	1	132	150	49	77	26	2.43	1.1	3.4	0.11	89.9
146	49	M	1.65	68	98	24.98	140	70	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	99	123			30	1.17	55	126	33	78	11	2.43	1.1	3.4	0.08	72.8
147	47	M	1.58	72	103	28.84	130	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	135	71			29	1.16	132	150	49	77	26	1.12	0.86	3.08	0.32	74.6
148	47	F	1.5	46	65	20.44	120	90	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	103	161			24	1.04	115	120	32	78	18	4.02	1.44	2.7	0.08	63.9
149	38	M	1.67	60	84	21.51	120	80	76	Y	N	N	N	N	N	N	N	Y	N	N	Y	N	N	Y	N			104		15	1.08	462	158	34	87	92	1.49	1.09	2.91	0.07	86.6
150	30	F	1.6	62	82	24.22	110	70	68	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N	145	200		7.18	23	1.17	155	217	42	135	31	2.37	0.98	3.02	0.07	62.5
151	42	M	1.65	76	98	27.92	110	70	72	N	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	108	102			22	1.38	107	144	34	89	21	1.77	1.35	3.53	0.09	62.9
152	50	F	1.6	80	108	31.25	140	90	82	N	N	N	N	N	N	N	N	N	N	N	y	N	N	N	N	168	192		6.5	32	1	143	170	43	95	43	29	1.08	1.79	0.19	65.6
153	45	M	1.65	68	98	24.98	160	110	88	N	N	Y	N	N	N	N	N	Y	N	N	Y	N	Y	N	N	168	308		8.8	40	1.9	97	170	32	107	19	3.5	1.21	1.07	0.38	41.6

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
154	40	M	1.64	88	108	32.72	150	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			98		27	1.1	175	128	29	66	35	3.47	1.49	3.77	0.07	83.5
155	50	M	1.46	75	102	35.18	120	80	88	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	130	170	311	11.5	69	1.6	189	141	24	85	38	150	0.3	10	0.53	49.5
156	26	M	1.66	76	94	27.58	120	80	90	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N			116		31	1.3	121	178	32	89	24	1.71	1.5	2.53	0.12	75.3
157	32	r	1.69	54	82	18.91	100	60	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	94	130			19	1	135	185	40	88	25	1.95	1.78	3.36	0.09	99.1
158	71	F	1.58	62	99	24.84	130	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	96	132			27	1.05	182	171	29	109	36	2.41	1.09	3.12	0.05	53.4
159	39	M	1.7	78	106	26.99	120	70	78	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	106	111			29	1.34	98	171	35	112	20	2.75	1.02	1.32	0.13	66.3
160	38	F	1.48	51	96	23.28	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	84	125		5.9	15	0.94	107	202	45	113	21	2.25	1.1	3.4	0.07	77
161	33	F	1.54	66	86	27.83	130	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			129		17	0.79	72	140	34	87	14	0.66	1.2	2.93	0.08	98.4
162	55	F	1.54	72	108	30.36	140	90	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			134	6.8	26	0.95	67	129	36	75	13	1.52	1.19	3.11	0.1	67.4
163	45	F	1.54	68	98	28.67	100	60	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			146		23	0.82	44	148	44	85	9	1.82	1.24	3.31	0.09	86.4
164	65	M	1.72	62	106	20.96	130	80	68	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N			98	4.5	27	1.2	95	152	47	86	19	1.52	1.29	3.28	0.09	63.1
165	48	M	1.55	56	86	23.31	120	80	88	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N			101	8.75	22	1.14	175	191	58	93	35	10.62	1.13	3.17	0.13	75.6
166	55	F	1.62	60	86	22.86	100	60	88	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	220	232	484	9.61	85	2.7	196	159	28	99	39	1.25	1.17	1.21	10.69	19.1
167	55	F	1.59	58	115	22.94	130	70	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	359	382		9.2	15	0.8	108	129	35	74	22	3.27	1.38	1.5	1.3	83
168	21	F	1.55	59	92	24.56	130	70	88	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			88		115	15	84	94	16	63	17	1.94	0.67	2.67	4	3
169	31	F	1.75	75	96	24.49	120	80	80	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	71	91			26	0.95	95	188	43	113	19	1.37	1.11	2.97	0.89	79.8
170	45	M	1.65	62	98	22.77	120	80	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	163	302		7.2	27	1.18	104	187	42	123	21	3.58	1.33	3.69	0.09	74.1
171	25	M	1.65	68	82	24.98	120	70	66	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	63	128			26	1.28	128	121	29	68	26	4.74	1.43	3.89	0.27	77.3
172	22	F	1.52	48	70	20.78	110	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			61		16	0.79	73	150	55	78	15	2.47	1.02	3.13	0.16	106.3
173	58	M	1.58	65	76	26.04	130	90	78	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	87	160			41	2.27	303	181	30	110	61	1.33	1.02	4.03	0.67	30.7
174	55	F	1.45	55	97	26.16	110	80	76	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	87	135			19	1.01	126	190	40	76	22	2.35	1.27	2.43	0.14	65.8
175	55	F	152	62	84	0.00	110	82	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	176	133		7.5	16	0.89	142	210	49	124	28	2.45	1.22	3.14	0.19	73
176	29	F	1.5	54	84	24.00	120	70	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			75		16	1	70	197	47	122	14	2.22	0.95	2.83	0.05	76.1
177	40	F	1.48	60	86	27.39	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	72	108			22	0.89	94	199	47	127	19	2.81	1.09	2.05	0.07	81.1
178	57	M	1.62	83	114	31.63	140	90	82	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y			102		18	1.08	225	144	28	82	45	3.32	1.11	3.47	0.09	75.8
179	51	F	1.54	86	114	36.26	130	80	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			64		19	1.02	90	133	37	84	18	2.92	1.05	2.83	0.14	63.6
180	37	F	1.58	96	121	38.46	130	90	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	164	162		6.8	20	0.93	77	175	39	110	15	0.78	1.25	2.65	0.09	78.5
181	44	M	1.7	91.5	106	31.66	120	90	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	91	109			24	1.56	169	196	39	121	34	2.25	1.05	3.7	0.05	53.2
182	64	M	1.56	75	111	30.82	150	100	86	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	91	148			18	1.17	128	215	46	130	26	4.24	1.1	3.17	0.09	65.5
183	43	M	1.65	68	102	24.98	170	100	84	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	74	127			21	1.24	114	148	29	95	23	2.29	1.58	3.59	0.14	70.8
184	49	M	1.62	76	108	28.96	110	70	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	218	350		8.5	19	0.93	163	236	55	140	33	0.84	1.17	3.39	1.36	96.1
185	28	F	1.56	62	96	25.48	110	70	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			84		22	0.87	78	174	38	108	16	1.2	1.11	2.91	0.08	90.7
186	48	F	1.62	78.5	100	29.91	110	60	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			79		16	0.85	40	157	45	99	8	0.76	1.21	2.9	0.07	81
187	49	M	1.69	80.8	106	28.29	100	60	82	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Y	Y	N			146		42	2.05	293	113	25	59	59	2.59	1	3.99	0.23	36.9
188	72	F	1.4	55	102	28.06	150	80	72	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			120		21	1.2	82	183	44	111	16	2.87	1.17	2.7	0.26	45.1
189	55	F	1.48	40	86	18.26	180	90	68	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			151	8.5	49	1.3	187	186	42	113	37	2.29	1.23	2.74	0.09	46.1
190	60	F	1.55	50	90	20.81	140	70	66	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			79	6.06	27	0.92	75	154	50	89	15	0.62	1.03	2.83	0.29	67.7
191	50	F	1.48	56	91	25.57	110	70	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	84	109		4.8	21	0.9	62	172	54	96	12	2.54	1.37	3.11	0.09	74.6
192	20	F	1.61	40	73	15.43	120	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			112		31	0.8	108	162	48	82	21	0.28	1.92	3.09	0.19	106.1
193	39	F	1.68	60	90	21.26	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			112		15	1	181	151	29	88	36	2.47	1.09	2.66	0.21	70.6
194	64	M	1.66	56	90	20.32	90	60	70	N	N	N	N	Y	N	Y	N	N	Y	Y	N	N	Y	N	N	72	155			28	1.24	77	101	29	62	15	1.96	1.28	3	0.97	61.1
195	59	F	1.46	44.2	90	20.74	110	70	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	154			22	0.87	112	151	42	93	22	2.54	1.14	3.44	0.19	72.9
196	69	F	1.62	78	106	29.72	100	70	86	Y	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N			148	8.5	104	2.4	142	101	36	60	28	1.72	1.52	1.78	1.3	19.9
197	53	M	1.7	70.5	96	24.39	110	70	82	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	77	221		7.2	19	1.1	265	206	38	119	53	4.43	1.37	2.74	0.11	76.2
198	52	M	1.61	60	88	23.15	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	74	93			18	1.04	51	105	32	60	10	3.94	1.13	3.24	0.1	82.2
199	68	M	1.67	67	97	24.02	140	90	78	N	N	Y	N	N	N	N	N	N	N	N	Y	Y	N	N	N	162	193		7.2	23	1.08	242	161	33	94	48	1.83	1.51	2.79	0.12	70.2
200	55	M	1.7	90	110	31.14	170	100	84	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	179	227		8.2	27	1.52	190	195	37	120	38	2.12	1.14	3.33	0.28	50.8
201	51	M	1.67	76	103	27.25	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	94	138			27	1.16	125	206	35	138	25	1.52	1.38	3.48	0.06	72.5
202	37	F	1.565	64	102	26.13	110	60	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	78	89			30	0.86	137	219	51	125	27	5.33	0.92	3.42	0.1	86.3
203	27	M	1.69	71	89	24.86	100	60	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	79	91			21	0.97	63	115	28	69	1.1	1.75	1.22	3.31	0.08	106.5
204	48	M	1.57	55	83	22.31	100	70	76	N	Y	Y	N	N	N	N	N	N	Y	Y	N	N	Y	Y	N			127	7.6	55	4.4	61	66	33	25	12	100	0.1	0.58	0.02	14.8

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
205	50	M	1.64	85	111	31.60	130	80	88	N	N	N	N	N	N	N	N	N	y	Y	N	N	N	N	N			100		28	1.6	99	180	43	110	20	6.73	1.12	2.88	0.2	49.5
206	65	M	1.6	75	110	29.30	150	90	76	N	Y	N	N	N	N	N	N	N	Y	Y	N	N	Y	Y	N	140	359		7.5	31	1.4	88	207	58	120	18	4.1	1.1	3.17	0.07	52.4
207	43	M	1.68	65.9	96	23.35	140	80	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N			92		18	0.93	79	139	30	87	16	1.58	1.53	3.23	0.19	100.2
208	37	F	1.48	60	94	27.39	120	80	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			127		17	0.87	116	172	43	101	23	2.41	1.15	3.54	0.3	85.1
209	53	M	1.72	78	110	26.37	140	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			92		31	1.13	67	168	39	106	13	2.99	1.12	3.3	0.06	73.8
210	33	M	1.72	68	96	22.99	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			105		18	0.94	102	147	31	90	20	2.55	1.19	3.88	0.05	106.1
211	41	F	1.53	81	109	34.60	120	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			101		16	0.8	72	184	37	122	14	3.49	1.09	2.98	0.04	91.6
212	50	M	1.64	55	76	20.45	140	80	78	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	112	258		7.6	104	5.5	64	154	38	30	13	2.26	1.12	1.91	9.05	11.1
213	34	M	1.63	59.5	84	22.39	110	70	88	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	Y	N			91		16	0.9	237	616	91	369	47	1.43	1.43	3.46	1.28	111
214	73	M	1.65	62.5	106	22.96	120	70	70	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			115		15	1.2	115	147	21	94	23	2.45	1.24	2.65	0.21	59.6
215	60	M	1.665	54.5	89	19.66	130	80	70	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	100	198		7.8	19	1.1	100	142	27	95	20	2.76	1.18	3.14	0.69	72.6
216	73	F	1.48	55	83	25.11	110	70	66	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			78		30	0.93	53	177	57	101	11	3.42	1.26	2.94	0.14	61
217	45	F	1.58	63.5	95	25.44	120	80	76	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	Y			100		21	0.93	69	135	39	81	14	0.84	1.62	3.14	0.05	74.2
218	45	M	1.635	66.5	98	24.88	120	80	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	139	162		7	19	1.17	240	119	26	64	48	1.55	1.14	3.01	0.11	74.8
219	20	M	1.6	66.5	86	25.98	120	80	62	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			71		17	1.06	102	105	35	58	20	3.29	1.59	3.73	0.09	100.5
220	28	M	1.635	64	90	23.94	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			105		41	0.92	43	108	41	55	9	1.07	1.55	4.04	0.11	112.8
221	43	M	1.58	68	104	27.24	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			97		18	1.03	89	140	38	83	18	2.45	1.13	3.46	0.07	88.6
222	61	M	1.7	74	108	25.61	130	80	82	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N			151	6.8	21	1.3	78	221	73	128	16	3.94	1.44	2.73	0.14	58.9
223	32	M	1.705	74.2	102	25.52	120	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N			101		16	0.81	310	271	48	171	62	2.13	0.97	2.26	0.19	117.6
224	40	M	1.605	67.5	102	26.20	120	80	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N			93		20	1.12	271	181	30	112	54	3.76	1.26	3.36	0.08	81.7
225	65	F	1.52	74	104	32.03	120	60	80	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	105	235		6.43	17	0.79	83	171	40	106	17	6.68	0.9	2.78	0.08	78.6
226	43	M	1.7	76	107	26.30	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	114	194		6.5	22	1.37	123	193	35	123	25	2.29	1.17	3.53	0.05	62.7
227	57	M	1.62	56	76	21.34	110	80	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	266	320		9	28	0.98	48	106	44	57	10	2.92	1.31	3.01	0.11	85.2
228	35	F	1.6	62	84	24.22	120	80	72	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	230	278		7.6	14	0.81	79	148	52	80	16	2.41	1.18	2.68	0.12	94.1
229	39	F	1.55	66	90	27.47	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			129		27	0.96	172	168	37	104	34	1.08	1.65	3.94	0.05	74.5
230	31	F	1.53	68	115	29.05	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	101	108			24	0.82	73	169	43	103	15	2.39	1.17	4.1	0.06	95.3
231	45	F	1.57	79.6	90	32.29	150	100	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	96	110			22	0.8	117	280	61	180	23	2.62	1.24	3.31	0.1	89
232	44	F	1.49	66.4	96	29.91	100	70	72	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	127	143			30	0.9	126	190	40	76	22	29.3	0.81	2.41	0.2	77.8
233	57	F	1.58	73.8	100	29.56	120	80	80	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	142	202		7.18	22	0.89	149	226	39	145	30	1.87	1.42	2.71	0.03	71.9
234	63	F	1.56	66.8	107	27.45	110	70	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	187	280		8.39	17	0.95	194	241	42	156	39	2.76	1.13	2.73	0.13	63.7
235	58	M	1.7	92.1	118	31.87	130	90	68	N	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	231	272		9.22	26	0.94	170	216	48	131	34	1.26	1.41	3.48	0.13	89
236	29	M	1.66	70.9	93	25.73	140	90	76	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N	Y	N	88	94			18	1.13	64	152	38	96	13	1.45	1.46	4.66	0.06	87.3
237	47	F	1.47	65.6	109	30.36	120	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	94	92			24	1.02	65	162	41	98	13	12	1.41	2.95	0.09	65.2
238	57	M	1.675	77.1	98	27.48	130	90	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	103	129			21	1.04	227	184	32	106	45	3.02	1.24	3.7	0.14	79.3
239	68	M	1.65	65.1	95	23.91	110	70	76	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	214	364			38	0.92	131	178	43	106	26	1.74	1.06	3.67	0.21	85.2
240	40	F	1.6	70.7	103	27.62	100	70	86	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	106	111			25	0.98	126	177	35	108	25	2.42	1.36	3.38	0.13	72.2
241	42	F	1.6	78.7	100	30.74	126	80	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	107	77			18	0.95	89	180	58	99	18	5.21	1.04	3.17	0.32	73.9
242	50	M	1.68	73.2	93	25.94	110	80	80	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N	274	439		7.42	24	1.14	151	191	45	111	30	1.93	1.28	3.71	0.08	74.6
243	20	F	1.42	40	72	19.84	130	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			96		31	1	83	103	29	64	17	2.24	1.16	3.06	0.6	81
244	46	F	1.515	44	79	19.17	150	90	80	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	80	123			37	1	333	278	43	160	67	2.47	1.08	2.34	0.11	67.5
245	46	F	1.5	64	82	28.44	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	305	540		10.5	24	0.9	85	117	30	67	17	2.46	1	3.23	0.12	76.7
246	52	F	1.5	65	101	28.89	140	100	76	N	Y	Y	N	N	Y	N	N	N	N	N	Y	N	N	N	N	225	217		10.1	37	1.5	266	282	52	175	53	3.19	1.21	2.02	0.5	39.6
247	70	F	1.46	58	108	27.21	110	60	76	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			201	8.5	41	1	241	210	36	127	48	2.38	1.05	4.44	0.23	57
248	48	F	1.5	68	101	30.22	140	80	82	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	87				22	0.9	153	184	37	111	31	3.81	1.31	2.8	0.4	75.6
249	20	F	1.45	42	74	19.98	120	80	78	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N			100		20	0.8	87	130	35	76	17	3.89	1.4	3.62	0.1	106.1
250	40	M	1.7	85	98	29.41	120	80	86	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	310	430		8.6	24	1.12	97	179	43	108	19	1.19	1.14	3.11	0.21	81.7
251	48	F	1.47	72	107	33.32	140	80	80	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N			95		17	0.8	80	169	45	101	16	2.01	0.87	3.62	0.17	87.2
252	50	M	1.55	52	76	21.64	110	80	88	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	64	107			15	1	195	142	26	83	39	2.39	1.21	3.56	0.16	86.2
253	76	M	1.55	60	91	24.97	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	92	141			28	1	190	154	29	92	38	18.93	0.799	3.33	0.22	72.8
254	57	M	1.58	76	104	30.44	170	110	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	134			37	1.7	237	190	35	115	47	1.41	1.21	2.83	1.29	43.8
255	51	F	1.55	69	93	28.72	110	80	72	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	90	112			31	1	56	147	40	84	11	3.16	1.27	2.8	0.08	65.2

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
256	42	M	1.67	84.2	100	30.19	120	80	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N				7.2	17	1.1	169	196	39	123	84	1.66	1.6	3.21	0.12	82.4
257	35	F	1.51	62.8	94	27.54	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			86		26	0.8	172	257	50	163	34	1.12	1.06	3.38	0.12	95.5
258	65	M	1.59	68.1	106	26.94	120	70	82	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	Y	N	227	336		7.5	17	1	192	157	34	91	38	1.02	1.3	2.84	0.15	78.6
259	52	M	1.64	73.1	94	27.18	140	100	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	112	152			15	1	65	172	41	108	13	4.51	1.46	3.09	0.01	86.2
260	46	M	1.64	61.6	88	22.90	170	100	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	85	141			21	1	124	200	41	124	25	1.59	1.08	3.39	0.16	89.9
261	52	M	1.61	50	80	19.29	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	192			23	0.7	105	143	33	88	21	1.09	0.98	3.16	0.28	108.5
262	23	M	1.695	59.6	83	20.74	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	95	116			15	1	63	135	60	66	13	3.16	1.27	2.6	0.1	105.6
263	73	M	1.67	71.4	101	25.60	140	70	80	N	Y	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N	105	144			25	1.1	62	157	47	92	12	2.79	1.43	3.49	0.1	66.2
264	28	F	1.59	60.1	79	23.77	120	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	92	107			18	0.8	55	148	40	91	11	2.76	1.16	3.14	0.09	100.3
265	49	M	1.71	73.2	91	25.03	120	86	78	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	134	233		7.8	26	1.23	240	203	43	116	48	2.93	1.22	3.49	0.08	68.5
266	31	M	1.69	73	90	25.56	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	111	220		5.8	26	1.08	59	226	49	145	12	3.93	1.04	2.71	0.09	91
267	56	M	1.64	70.3	96	26.14	120	90	82	N	Y	Y	N	N	N	N	N	Y	Y	N	N	N	Y	Y	N	123	303		8.3	27	1.81	510	147	28	78	102	4.7	1.04	2.9	0.44	40.9
268	29	F	1.649	49	75	18.02	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			99		24	0.9	126	175	35	84	15	2.16	1.78	3.1	0.08	86.4
269	67	F	1.5	62	106	27.56	160	90	76	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	249	346		7.3	23	1.2	191	118	38	59	38	1.91	1.8	3.64	0.15	46.7
270	54	M	1.62	96	88	36.58	120	80	78	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	93	165			32	1.1	132	237	52	143	26	5.42	1.32	4.22	0.1	75.7
271	60	F	1.56	60	98	24.65	110	70	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	105	114			30	0.9	108	219	53	137	22	9.07	1.03	3.03	0.09	69.5
272	33	M	1.72	76	92	25.69	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	89	112			28	1.13	177	162	30	102	35	1.81	1.1	3.78	0.06	84.9
273	37	M	1.68	74	88	26.22	120	60	74	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	93	101			16	1.32	49	108	39	63	10	8.37	1.02	2.84	0.1	68.4
274	31	M	1.6	46	75	17.97	110	80	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			129		33	1.12	80	142	32	85	16	9.787	1.12	3.46	0.26	87.1
275	56	M	1.65	76	105	27.92	120	80	72	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	124	181		7.2	25	1.64	84	183	37	111	17	1.79	1.18	4.9	0.07	46.1
276	44	M	1.62	70	93	26.67	130	80	68	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	98	191		7.5	38	1.27	96	203	41	121	19	13.86	0.71	3.72	0.07	68.3
277	47	F	1.41	56	94	28.17	80	60	76	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N			94		21	0.94	135	106	25	55	27	25.97	0.79	3.57	0.36	72.2
278	50	F	1.58	65	89	26.04	120	90	70	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N		`	87		21	0.91	95	169	38	98	19	2.95	1.35	3.92	0.003	73.6
279	45	F	1.65	65	103	23.88	120	80	78	N	N	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	144	253		7.4	17	0.75	147	270	40	175	29	4.38	0.92	2.75	0.002	96.3
280	67	F	1.53	62	100	26.49	120	90	68	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	95	139		4.51	20	0.92	141	223	49	130	28	1.2	1.17	4.03	0.004	64.4
281	31	M	1.67	71	86	25.46	120	80	74	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	107	126			14	1.08	184	144	23	84	37	0.74	1.58	3.74	0.002	91
282	50	M	1.7	108	110	37.37	120	80	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	207	193		8.4	24	1.05	176	188	29	113	35	3.58	1.1	3.35	0.001	82.4
283	27	M	1.65	68.5	84	25.16	120	60	78	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N			147		16	0.86	381	257	33	154	76	0.89	1.15	3.55	0.07	113.9
284	52	F	1.5	58	94	25.78	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	93	106	160		15	1	126	162	35	111	26	3.24	0.96	3.32	0.09	64.7
285	37	F	1.4	49	83	25.00	110	70	76	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N			115		15	0.8	88	182	54	95	18	3.88	1.52	3.28	0.002	94.2
286	39	F	1.64	93	106	34.58	140	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	272	350		9	22	0.9	78	137	35	90	16	1.28	1.39	3.64	0.005	80.5
287	68	F	1.65	80	106	29.38	120	80	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			130		20	1	216	182	38	104	43	1.57	1.08	2.08	0.12	57.8
288	50	F	1.52	49.5	84	21.42	140	90	82	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	396	575		9.8	15	0.8	110	278	61	178	22	3.36	1.35	3.39	0.02	86
289	28	F	1.595	51	82	20.05	100	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			92		15	0.2	68	121	31	65	14	12.82	0.8	3.4	0.1	78.1
290	55	F	1.562	74	99	30.33	120	70	82	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N			120		24	1	85	179	43	103	17	3.43	1.04	3.17	0.13	70.4
291	50	F	1.6	65.5	95	25.59	140	90	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			99		27	0.9	244	231	42	128	49	0.6	1.24	3.68	0.1	74.6
292	50	F	1.59	80	106	31.64	160	100	78	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			118		78	7.5	91	129	41	76	18	3.94	1.04	2.98	1.95	5.7
293	43	F	1.64	48	77	17.85	120	60	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			105		21	0.9	56	150	37	86	11	2.23	1.35	3.52	0.1	78.3
294	60	F	1.7	54	79	18.69	110	70	80	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			106		29	1	103	188	56	109	21	1.02	1.3	2.84	0.09	61.2
295	50	F	1.56	60	96	24.65	130	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			82		17	0.9	107	202	48	113	21	6.66	1.13	3.37	0.13	74.6
296	50	F	1.58	65	98	26.04	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	77	81			22	0.8	123	226	47	141	25	16.02	0.97	3.05	0.2	86
297	53	M	1.68	79.5	98	28.17	130	80	78	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	N	N	103	190			49	1.56	214	123	15	76	43	2.2	1.53	3.3	0.5	50
298	65	F	1.62	74	100	28.20	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	100	146			17	1	143	169	48	90	29	1.65	1.31	4.29	0.09	59.1
299	30	M	1.6	65	88	25.39	110	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	93	96			27	1.11	85	161	36	92	17	1.83	1.31	4.29	0.02	88.6
300	32	M	1.605	76.2	97	29.58	110	70	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	126			19	0.97	81	152	35	91	16	3.67	1.21	3.75	0.05	102.9
301	60	F	1.51	48	83	21.05	110	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	83			16	0.84	126	203	51	107	25	11.36	0.8	3.38	0.77	75.5
302	52	M	1.6	65	92	25.39	130	80	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	108	108			25	1.16	81	198	40	115	16	0.99	1.06	3.23	0.12	72
303	44	F	1.59	90	104	35.60	120	80	78	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	89	126			22	1.01	105	189	40	117	21	1.63	1.44	3.3	0.13	67.6
304	51	M	1.6	70	76	27.34	170	100	74	N	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	106	163			24	1.03	124	129	30	70	25	3.1	1.33	2.95	0.2	83.7
305	50	F	1.44	52	72	25.08	160	100	72	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	223	348		8.8	32	1.23	158	277	65	159	32	1.67	1.34	3.21	0.59	51.1
306	34	F	1.52	52	74	22.51	110	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	94	97			26	0.9	66	127	62	55	13	2.28	1.13	3.04	0.68	80.4

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
307	63	M	1.6	72	84	28.13	150	80	86	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	74	133			36	1	78	183	47	103	16	3.7	1.22	3.52	0.12	79.7
308	46	M	1.608	64	94	24.75	120	70	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	266	269		10.1	23	1	215	213	35	129	43	2.55	1.11	3.57	0.24	89.9
309	45	M	1.58	64	82	25.64	110	80	76	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	N			95		26	1.1	141	194	44	115	28	4.47	1.06	4.03	0.09	80.6
310	44	M	1.56	84	107	34.52	130	40	82	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N	104	151			37	1.8	215	133	26	86	43	8.16	1.55	3.47	0.21	44.8
311	65	M	1.65	76	102	27.92	120	80	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	95	113			24	0.98	105	179	46	100	21	176	1.08	3.19	0.28	80.6
312	29	M	1.72	76.5	86	25.86	110	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	102	116			20	1.1	106	166	38	96	21	0.85	1.38	4.29	0.15	90.2
313	45	M	1.56	41.5	77	17.05	100	60	78	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N			145		35	1.5	72	92	44	49	14	4.47	1.06	4.03	2.14	55.4
314	47	M	1.7	63.5	85	21.97	150	100	68	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N			84		21	1.1	150	156	33	95	30	3.7	1.22	3.55	0.1	79.5
315	52	M	1.68	59.5	94	21.08	150	90	80	N	N	Y	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	169	562		8.6	27	1.8	106	125	51	56	21	4.03	1.26	4.17	1.3	42.3
316	70	M	1.58	55	85	22.03	110	70	90	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N			83	5.8	45	2.9	80	115	33	66	16	3.33	1.03	2.41	1.3	21
317	43	M	1.575	45	79	18.14	110	70	90	N	Y	N	N	N	N	N	N	Y	N	N	Y	N	Y	Y	N			100		26	1	76	127	36	76	15	1.61	1.21	1.76	0.07	91.8
318	51	F	1.6	74	101	28.91	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	82	88			25	0.7	79	185	53	115	18	2.27	1.27	3.55	0.26	101
319	48	F	1.62	65	102	24.77	110	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	92			22	1.16	126	166	34	115	25	0.68	1.18	3.2	0.13	55.4
320	59	M	1.72	76	99	25.69	120	80	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	79	152			23	1.4	82	72	46	31	12	2.99	1.25	3.37	1.27	54.6
321	55	M	1.68	62	97.5	21.97	110	80	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	213	361		8.2	22	1.02	229	141	30	83	46	1.74	1.38	3.17	0.29	82.4
322	53	F	1.5	43.8	79	19.47	140	80	78	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	138	281		5.1	38	18	81	94	35	52	16	3.55	1.41	3.34	1.82	31.6
323	60	M	1.46	57.4	96	26.93	180	100	86	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			118		32	1.9	74	103	85	65	15	4.3	1.23	3.02	0.4	37.5
324	45	F	1.51	59.6	87	26.14	130	90	76	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	114			21	0.9	346	268	54	156	69	4.07	0.96	1.94	8.9	77.2
325	33	M	1.615	75.1	94	28.79	110	80	72	N	N	N	N	N	N	N	Y	N	N	N	Y	N	Y	N	N	83	130			25	1.8	100	123	43	74	20	2.08	1.24	3.19	0.1	48.4
326	48	M	1.565	65.8	95	26.87	100	60	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	106	129			59	2.2	132	170	49	95	26	2.47	1.37	2.99	0.25	34.2
327	55	F	1.412	33.5	68	16.80	130	80	78	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	111	107		4.1	29	1.3	159	134	34	81	32	7.17	0.93	3.02	0.58	46.1
328	42	M	1.65	66	99.5	24.24	110	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	61	87			21	1.33	223	166	32	103	45	1.29	1.38	3.11	0.87	65.5
329	42	M	1.64	76	98	28.26	120	80	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	62	72			18	1.05	113	151	36	101	23	1.53	1.3	3.16	0.08	87.1
330	45	F	1.56	59.5	92	24.45	120	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	71	117			14	0.81	69	161	50	97	14	3.57	1.07	3.5	0.17	87.7
331	33	F	1.58	100	112	40.06	120	80	76	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	71	105			17	0.86	120	213	48	140	24	0.03	1.82	4.92	0.12	88.8
332	36	M	1.595	57.5	88	22.60	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	70	100			18	1.15	165	160	39	99	33	2.43	1.27	2.7	0.02	81.4
333	39	M	1.72	74	98	25.01	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	93	113			23	0.97	51	141	42	95	10	16.19	0.95	2.86	0.06	97.9
334	62	M	1.63	70	95	26.35	120	70	84	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	81	112			17	1.19	89	132	40	89	15	0.91	1.24	2.71	0.09	65.1
335	52	F	1.51	62	103	27.19	130	80	78	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	264	764		9.5	34	0.94	587	358	60	192	117	1.29	1.38	2.72	0.32	69.7
336	70	M	1.57	58	83	23.53	120	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	85	125			28	1.5	120	129	28	83	24	2.12	1.11	2.28	0.1	46.5
337	47	F	1.52	67	101	29.00	120	80	78	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N			98		21	0.73	124	154	36	99	25	6.65	1.53	3.02	0.13	98.1
338	56	M	1.65	78	102	28.65	110	70	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N			196		26	0.95	124	175	41	113	25	4.85	1.19	2.62	0.08	89.1
339	43	F	1.62	60	98	22.86	110	80	82	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	76	68			24	0.8	63	146	44	90	13	3.48	1.53	2.92	0.08	90.3
340	38	F	1.57	73.2	96	29.70	140	90	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			93		17	0.78	47	148	46	93	9	1.05	1.35	2.98	0.09	96.4
341	46	M	1.74	62.5	80	20.64	100	50	70	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	N	72	69		4.3	17	0.98	33	123	49	77	7	1.57	1.27	2.55	0.13	92.1
342	39	M	1.625	74.9	103	28.36	110	60	88	N	Y	N	N	N	N	N	N	Y	N	N	Y	N	N	Y	N	67	86		5.15	22	1.1	72	100	24	75	14	0.51	1.65	2.56	0.11	84.1
343	51	M	1.72	80	102	27.04	120	70	78	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	100	205		7.5	25	0.89	80	169	40	112	16	9.04	1.12	2.56	0.11	99
344	50	M	1.77	78.6	98	25.09	140	90	82	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	80	67		3.87	18	0.98	159	134	34	81	32	3.27	1.51	3	0.2	89.5
345	47	F	1.58	70	99	28.04	110	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	122	178			23	0.92	104	140	32	99	21	1.89	1.16	2.4	0.09	74.1
346	49	M	1.72	76	98	25.69	110	70	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	79	135			24	1.12	48	122	30	90	10	2.7	1.32	3	0.06	76.7
347	50	F	1.62	15.2	103	5.79	110	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	102	117			15	0.77	221	257	68	159	44	3.62	1.36	2.2	0.15	90
348	55	M	1.72	68	90	22.99	110	60	78	N	N	Y	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	67	199	259	6.6	34	1.6	202	170	35	103	40	3.43	1.25	2.62	0.16	47.8
349	75	M	1.6	53	93	20.70	150	80	82	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	102	210		5.9	28	1.3	80	126	29	87	16	4.49	1.19	2.5	1.54	53.4
350	44	M	1.67	73	104	26.18	120	80	80	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	y	N	255	349		8.1	21	0.8	124	208	47	140	25	2.1	0.92	2.85	0.18	108.6
351	62	M	1.53	70	113	29.90	130	80	76	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N			96	7.2	64	3.2	116	144	32	89	23	3.33	1.03	2.41	2.6	19.7
352	60	M	1.44	46	80	22.18	130	70	74	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	148	220		6	53	2	184	128	21	73	37	3.82	1.03	2.78	1.75	35.2
353	60	M	1.62	76	105	28.96	160	100	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	76	107	164	4.8	40	1.1	169	186	40	124	34	0.62	1.46	2.04	0.2	72.6
354	61	M	1.62	72	98	27.43	100	70	96	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			127		37	1.1	100	137	24	94	20	1.2	1.43	2.77	0.23	72.1
355	52	M	1.59	59.5	100	23.54	140	80	78	N	Y	Y	Y	N	N	N	N	N	Y	N	N	N	N	Y	N	150	316		8.5	83	3.2	224	164	33	96	45	16.07	0.59	2.9	3.3	21.1
356	55	M	1.65	84.5	115	31.04	110	80	80	N	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N			250	7.2	20	1	224	203	50	123	45	6.96	1.26	1.26	0.17	84.4
357	61	F	1.55	58.5	100	24.35	110	80	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	135	220		7.2	22	0.82	155	237	53	156	31	2.29	1.17	3.05	0.23	77.2

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
358	38	M	1.61	62.8	95	24.23	180	70	80	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	Y	N	84	123			33	1.72	259	143	31	83	52	100	0.53	1.94	0.19	49.3
359	27	M	1.7	70	88	24.22	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	86	101			14	1.24	115	137	32	94	23	4.55	1.48	3.71	0.16	79.2
360	65	M	1.59	59.1	88	23.38	110	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	80	113			24	1.04	155	131	27	89	31	3.14	1.24	2.89	0.07	75
361	51	F	1.525	63.2	102	27.18	110	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	90	162			15	0.79	177	141	38	85	35	3.12	1.02	3.09	0.17	86.7
362	39	M	1.6	58.2	94	22.73	200	120	80	Y	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	163	292		7.8	21	1.19	120	215	46	155	24	2.51	1.37	2.76	4.05	76.5
363	21	M	1.64	49	78	18.22	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	74	73			15	0.94	82	122	44	72	16	1.11	1.78	4.5	0.13	115.4
364	55	M	1.65	61	90	22.41	150	100	78	N	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	172	288		6.9	73	3.2	118	177	29	127	24	1.89	1.31	2.1	8.8	20.7
365	33	M	1.7	90.1	94	31.18	120	90	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	78	144			22	1	85	204	52	138	17	6.26	1.12	3.97	0.06	98.5
366	54	F	1.53	70.6	104	30.16	130	90	80	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	70	89			15	1	115	226	48	157	23	7.99	1.08	2.93	0.07	63.8
367	45	F	1.55	58	98	24.14	110	70	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			58		30	0.9	87	223	64	136	17	1.19	1.16	2.91	0.05	77.2
368	40	F	1.52	55	90	23.81	170	100	70	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	73	81		4.4	21	0.6	59	133	54	75	12	1.43	1.49	3.52	0.13	114
369	42	F	1.5	52.5	85	23.33	110	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			85		19	1	74	169	54	106	15	1.41	1.2	2.92	0.05	69.4
370	21	F	1.585	41	68	16.32	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			69		16	0.6	72	124	28	75	14	0.38	1.56	1.86	0.8	130.3
371	43	F	1.67	82.5	103	29.58	110	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	114	169			27	0.9	163	216	54	138	33	2.87	1.13	2.11	0.12	78.3
372	60	F	1.538	36.5	73	15.43	100	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			83		19	0.6	161	178	32	121	32	4.41	1.08	2.76	0.21	99.1
373	39	M	1.62	69.6	93	26.52	100	70	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	69	110			25	1.35	105	174	38	114	21	1.24	1.21	3.02	0.11	65.7
374	42	M	1.838	78.2	94	23.15	110	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	91	129			27	1.06	109	126	32	74	22	1.24	1.24	3.02	0.15	86.1
375	65	M	1.68	65	96	23.03	110	60	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	129			19	0.9	68	203	52	131	14	3.02	1.06	2.78	0.1	89.3
376	50	M	1.602	80.3	102	31.29	110	80	84	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	151	95		6.8	21	1.12	85	143	47	85	17	2.3	1.44	3.81	0.14	76.2
377	50	M	1.67	73.6	101	26.39	90	60	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	N	206	175		7.8	20	1.12	187	179	34	117	37	0.93	1.17	3.25	0.08	76.2
378	50	M	1.62	78.2	102	29.80	110	70	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	115	251		7.2	26	1.01	152	128	32	81	30	3.7	1.2	3.22	0.13	86.3
379	52	M	1.725	88.6	110	29.78	120	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			84		21	1.2	97	214	40	143	19	4.03	1.27	3.43	0.2	69.1
380	55	M	1.56	75	108	30.82	130	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	137			24	0.1	76	156	37	102	15	1.22	1.38	4.01	0.19	95.8
381	38	M	1.695	73.8	98	25.69	120	90	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	85	168			17	1.08	140	214	46	136	28	4.94	1.28	3.9	0.15	86.6
382	53	M	1.565	63.8	92	26.05	130	80	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	89	110			15	0.8	81	149	52	81	16	2.4	0.98	3.8	0.09	102
383	72	M	1.65	80	104	29.38	120	80	80	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	85	96			22	1.24	95	166	37	111	19	3.61	1.11	3.7	0.11	60.1
384	53	M	1.592	56.3	88	22.21	100	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	80	115			25	1	79	188	45	125	16	2.73	1.11	4.43	0.13	85.5
385	50	M	1.625	62	92	23.48	110	70	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	67	123			38	1.31	92	190	53	127	18	1.82	1.29	3.62	0.1	63
386	34	F	1.65	68	86	24.98	100	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	64	95			18	0.82	48	119	40	70	10	3.7	1.23	4.24	0.1	93.4
387	52	F	1.59	68	102	26.90	110	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N		103	150			39	0.98	77	133	45	80	15	3.24	1.35	3.53	0.1	66.3
388	61	F	1.41	76	108	38.23	120	90	80	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	94	191		9.9	45	1.2	179	151	40	92	36	5.45	1.59	0.93	3.48	48.7
389	45	M	1.54	74	102	31.20	120	80	80	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	240	398		7.4	20	0.97	298	189	36	116	60	1.95	1.21	3.54	0.26	93.9
390	32	M	1.68	72.6	84	25.72	120	80	74	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	78	150			23	1.01	155	200	44	130	31	1.94	1.06	4.05	0.07	98
391	79	M	1.635	68	88	25.44	190	90	72	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N			106		31	1.2	62	157	29	101	12	1.03	1.16	4.07	5.08	57.2
392	60	M	1.675	55	79	19.60	120	90	78	N	Y	N	Y	N	N	N	N	N	Y	N	N	N	N	N	N			111		208	11.3	92	190	53	127	18	2.74	1.33	1.61	4.28	4.3
393	63	M	1.54	50	72	21.08	100	60	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			90		49	0.9	67	110	32	67	13	3.12	1.35	2.79	0.2	90.6
394	40	M	1.6	59	86	23.05	110	80	84	N	N	N	N	N	N	N	N	Y	N	N	Y	N	Y	N					65		28	1.1	63	127	45	65	13	0.708	1.91	3.1	0.16	83.5
395	59	F	1.54	54	78	22.77	120	90	76	N	Y	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N			86	4.9	22	0.6	153	134	37	79	31	3.19	1.11	1.94	0.2	99.8
396	55	M	1.65	67	104	24.61	180	110	76	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	114	160	284	7.31	33	1.4	190	139	29	88	38	2.44	1.34	2.27	0.31	56.2
397	61	M	1.58	56	76	22.43	170	100	78	N	Y	N	N	Y	N	Y	N	N	N	N	Y	N	N	N	N			91		27	1.1	84	163	39	111	17	3.28	1.22	2.5	0.01	72.1
398	56	M	1.6	72	105	28.13	130	80	80	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N	218	134	195	7.2	27	1.1	115	67	22	60	23	5.02	1.15	3.42	0.32	74.6
399	74	M	1.62	54	78	20.58	120	80	76	N	N	N	N	Y	N	N	N	N	Y	Y	N	N	Y	N	N			80	4.4	66	1.4	109	143	36	96	22	3.76	1.91	3.34	0.04	49.1
400	50	M	1.73	58	78	19.38	120	70	78	N	N	N	N	Y	N	N	N	N	N	N	Y	Y	Y	N	N			77	4.98	15	1.2	93	122	30	66	19	3.11	0.94	3.56	0.22	70.1
401	69	M	1.52	64	102	27.70	100	60	78	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	124			5.88	76	2.76	122	135	29	108	24	2.5	1.27	3.06	0.26	22.4
402	36	M	1.65	76.3	95	28.03	130	90	86	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N			110		26	1.1	103	162	41	95	21	2.27	1.27	3.55	0.06	85.9
403	57	M	1.65	63	94	23.14	120	84	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	79	107			16	1.16	110	114	31	86	22	1.68	1.31	3.51	0.12	6.95
404	38	M	1.67	71.3	93	25.57	180	110	80	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	88	108			20	1.38	232	130	37	88	46	2.78	1.34	3.51	0.27	64.4
405	55	M	1.65	72.7	96	26.70	120	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	81	90			18	1.25	115	124	50	79	23	3.73	1.39	3.54	0.09	64.4
406	58	F	1.46	68	94	31.90	120	80	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	79	74			16	0.88	207	165	48	116	41	3.1	0.95	2.93	0.06	83.3
407	50	F	1.595	65.1	104	25.59	130	80	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	120			18	0.87	251	117	29	77	50	2.92	1.11	3.26	0.05	77.7
408	47	F	1.59	51	90	20.17	130	70	80	N	N	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	106	228			22	0.8	291	160	45	78	58	2.47	1.09	3.3	0.08	87.8

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
409	68	M	1.57	52	78	21.10	130	80	84	N	N	N	N	Y	N	N	Y	N	Y	N	N	N	N	N	N			81	4.35	36	1.31	103	105	30	57	21	3.25	2.33	2.95	0.1	55.5
410	50	M	1.65	53	84	19.47	150	90	74	N	N	Y	N	Y	N	N	N	N	Y	Y	N	N	Y	Y	N	186	288		8.6	27	1.4	330	182	42	110	66	9.72	1.43	2.99	0.87	63
411	62	F	1.58	48	94	19.23	140	100	78	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	257	420	254	8.69	17	0.92	214	138	37	71	43	2.96	1.02	2.24	0.1	66.7
412	58	M	1.7	71	88	24.57	120	70	84	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	255	260		7.7	18	1.24	118	123	28	73	24	2.57	1.53	2.86	0.14	63.7
413	63	M	1.623	78.6	96	29.84	140	90	82	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	169	175		6.1	17	1.09	106	177	35	108	21	8.34	1.05	3.18	0.15	71.9
414	70	M	1.68	75.2	94	26.64	110	70	82	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	188	424		7.2	19	1.17	136	172	33	104	27	1.85	1.56	2.99	0.25	62.8
415	52	M	1.57	75.2	109	30.51	110	80	76	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	123	217		6.3	28	1.12	77	195	43	120	15	1.78	1.44	3.72	0.07	75.1
416	40	F	1.53	80	115	34.17	120	80	76	N	N	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	416	508		10.4	16	0.9	128	203	32	130	26	9.22	1.38	2.57	0.16	80
417	50	F	1.55	57	88	23.73	100	70	68	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N			104	7.6	17	0.92	68	108	24	64	14	3.05	1.25	2.51	0.06	96.6
418	50	F	1.53	53	95	22.64	130	70	70	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N	75	121			19	0.9	85	160	31	100	18	2.94	1.09	2.44	0.39	74.6
419	31	M	1.62	44	74	16.77	110	70	60	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	Y	N			104		32	1.1	46	81	23	48	9	1.69	1.5	2.84	0.27	89
420	68	M	1.56	48	82	19.72	110	80	82	N	N	N	N	Y	N	Y	N	N	N	N	Y	N	N	N	N			91	5.1	33	1.3	147	90	25	55	29	62	0.8	3.51	0.16	56.1
421	42	M	1.74	70.5	102	23.29	110	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	83	121			23	1.4	49	134	44	80	10	2.57	1.58	2.9	0.06	61.5
422	59	F	1.57	65	106	26.37	160	100	78	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	392	398		8.74	22	1	202	178	26	110	40	0.58	2.09	3.31	0.52	23.8
423	74	M	1.6	66	85	25.78	110	90	80	N	N	N	N	Y	N	N	N	N	N	N	Y	N	Y	Y	N			74		21	1	162	124	31	72	32	1.21	1.27	3.37	0.05	97.8
424	70	F	1.54	75	110	31.62	110	70	80	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	116	133		6.2	36	1.4	150	209	38	123	30	0.38	1.57	1.71	3.4	38
425	39	F	1.69	58	84	20.31	110	70	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			125		40	0.9	198	177	15	102	40	2.53	1.05	2.9	0.35	80.5
426	43	F	1.663	64	91	23.14	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			77		21	0.9	76	191	37	115	15	2.17	1.36	3.48	0.09	78.3
427	67	M	1.72	78	91	26.37	130	90	78	N	Y	Y	N	Y	N	N	N	Y	N	N	Y	N	N	N	N	171	320	293	5.7	29	1.1	157	142	21	97	24	5.65	1.21	2.31	0.62	68.1
428	45	M	1.38	47.5	85	24.94	110	80	82	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	89	223		7.2	117	3.4	85	161	36	92	17	7.61	1.4	2.75	0.62	20.6
429	35	M	1.45	43	75	20.45	100	60	86	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	N	N			88		18	1.1	67	82	21	46	13	2.91	1.68	3.18	0.22	86.5
430	49	M	1.72	55	76	18.59	100	60	82	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	152	93		5.8	17	1.19	67	89	28	44	13	2.83	1.31	3.07	0.05	71.3
431	42	F	1.6	85	104	33.20	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	83	137			29	0.78	60	125	36	69	12	0.02	4.66	10.13	0.17	93.8
432	55	M	1.56	44	87	18.08	120	80	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	187	467		8.1	81	3.3	110	125	27	75	22	0.694	1.79	1.67	1.18	19.9
433	55	M	1.69	57	86	19.96	120	80	82	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			62		23	1.3	143	169	48	90	29	2.71	1.14	3.51	0.31	61.4
434	40	M	1.83	75	90	22.40	150	100	72	N	Y	N	N	N	N	N	N	Y	N	N	Y	N	Y	Y	N			113		22	0.9	87	140	38	88	17	0.948	1.43	1.79	0.14	106.5
435	62	M	1.65	88	102	32.32	160	100	70	N	Y	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	104	143		7.3	145	137	93	86	37	38	19	1.3	1.92	1.07	0.42	3.4
436	75	M	1.58	70	87	28.04	130	70	78	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	230	335		9.1	36	2.2	211	137	29	87	42	1.05	1.27	2.02	4.7	27.7
437	45	M	1.61	90	119	34.72	110	70	80	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N			97		27	1.1	158	277	65	159	32	3.52	1.09	3.12	0.16	80.6
438	40	F	1.53	68.2	91	29.13	110	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N			97	4.72	19	0.88	159	147	32	87	32	3.37	1.2	2.63	0.08	82.2
439	67	F	1.49	64.1	95	28.87	120	70	76	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N			82		31	0.87	62	200	53	118	12	1.11	1.58	2.69	0.07	68.9
440	27	F	1.49	64.1	95	28.87	90	50	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	89	97			16	0.96	63	151	36	92	13	3.52	1.21	2.69	0.07	81.1
441	50	M	1.755	83.1	105	26.98	160	110	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	75	86			31	1.08	276	181	27	105	55	3.11	1.56	2.72	0.14	79.6
442	55	M	1.642	66.2	93	24.55	140	80	62	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	87	112			21	1.2	154	197	46	118	31	2.6	1.22	2.89	0.11	67.7
443	40	M	1.67	77.3	97	27.72	110	80	72	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	77	110			19	1.23	97	169	31	109	19	5.56	1.48	2.68	0.04	73
444	40	F	1.49	66	98	29.73	150	100	78	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	178	258		6.5	15	0.8	271	173	29	98	54	0.18	3.82	9.14	0.2	92.2
445	36	F	1.55	65	98	27.06	130	70	78	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N			83		15	0.8	88	139	27	84	18	2.64	1.48	2.57	0.09	94.8
446	62	F	1.4	78	127	39.80	130	80	76	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	96	176			39	1.8	157	93	27	47	31	3.77	1.5	2.3	2.39	29.6
447	31	M	1.642	61.9	85	22.96	110	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	85	112			16	1.02	121	202	46	122	24	3.55	1.36	1.02	0.24	97.5
448	58	M	1.58	72.5	98	29.04	120	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	91	120			18	1.09	126	132	27	86	25	3.59	1.16	3.2	0.3	74.4
449	25	F	1.58	83.6	104	33.49	100	70	90	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	83	71			17	0.9	65	156	42	96	13	2.6	1.2	2.35	0.13	88.9
450	72	M	1.64	75.8	106	28.18	120	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	108	122			28	1.18	110	149	35	91	22	3.66	1.15	3.09	0.34	64.5
451	37	F	1.54	63.9	95	26.94	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	67	92			16	1.01	154	216	42	133	31	4.25	1.1	2.84	0.09	71.1
452	53	M	1.708	77.9	101	26.70	110	80	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	82	84			35	0.97	89	122	46	72	18	0.02	1.19	3.93	0.09	88.8
453	48	F	1.572	79.4	101	32.13	120	70	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	100			15	0.72	100	139	42	83	20	2.21	1.17	3.4	0.12	99
454	43	M	1.68	63.5	86	22.50	90	50	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	80	93			18	1.17	132	164	33	103	26	2.52	1.38	3.42	0.15	75.9
455	63	M	1.65	77.4	104	28.43	150	100	86	N	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	101	124			28	1.24	147	217	40	143	29	4.1	0.88	3.33	0.13	61.5
456	53	F	1.55	98.3	114	40.92	130	80	82	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	85	115			24	1.03	118	161	41	97	24	1.33	1.27	3.16	0.08	62
457	84	M	1.72	45	88	15.21	130	90	82	N	N	N	N	Y	N	N	N	N	Y	N	N	N	N	N	N			89	5.51	83	2.4	181	87	32	47	36	2.24	1.55	6.39	0.32	23.9
458	63	M	1.75	70	100	22.86	180	110	78	N	Y	N	N	Y	N	N	N	N	N	Y	N	N	Y	Y	N			96	5.2	30	1.34	380	190	46	115	76	17.75	0.5	2.17	0.04	56
459	60	M	1.71	53	88	18.13	90	70	88	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	Y	N	92	125		7	54	1.3	68	86	11	55	14	89.19	0.49	1.82	0.11	59.3

S.No	Age.Yr	Sex	Ht(m)	Wt(Kg)	WC(cm)	BMI	SBP	DBP	PR/min	Nativemedicine (Y/N)	HT(Y/N)	DM(Y/N)	HYPOTHYROID(Y?N)	MI(Y/N)	CARDIACVALVULARILLNESS	SURGERY	RENALCALCULI	CVA	Farmer(Y/N)	Agrochemexposure(Y/N)	Nonfamer(Y/N)	Dye/Toxinexposure(Y/N)	Smoking(Y/N)	Alcohol(Y/N)	F.H.Renaldis.(Y/N)	FBS(mg/dl)	PPBS(mg/dl)	RBS(mg/dl)	HbA1C(%)	B.Urea(mg/dl)	S.Creatinine(mg/dl)	TGL(mg/dl)	TC(mg/dl)	HDLc(mg/dl)	LDLc(mg/dl)	VLDL(mg/dl)	TSH(µU/L)	FreeT4(pg/ml)	FreeT3(ng/ml)	UrinespotPCR	eGFR
460	38	M	1.6	58	88	22.66	100	80	100	N	N	N	N	Y	N	N	N	N	N	N	Y	N	Y	N	N			131		41	1.2	160	171	24	103	33	3.5	1.4	2.66	0.18	76.2
461	60	F	1.58	68	98.5	27.24	110	70	72	N	N	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	87	94	134	5.35	20	1.3	287	132	36	74	57	33.32	0.93	2.53	0.13	44.6
462	62	F	1.5	47	76	20.89	150	90	86	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N			147		34	0.8	249	138	51	80	50	2.1	1.91	2.46	0.21	79
463	51	M	1.67	69.5	100	24.92	120	80	82	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	214	238		7.39	28	0.9	150	218	47	138	30	1.83	1.35	3.09	0.08	98.5
464	47	M	1.672	75.1	92	26.86	120	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	235	299		7.4	36	1.1	145	188	36	120	29	6.48	1.27	3.26	0.58	79.5
465	42	M	1.625	72.2	99	27.34	120	80	82	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	179	333		7.85	21	1.1	146	197	39	132	29	2.32	1.38	3.38	0.25	82.4
466	56	M	1.645	83.3	104	30.78	140	90	88	N	Y	N	N	N	N	N	Y	N	N	N	Y	N	N	N	N	102	138			17	1.2	144	169	37	98	29	0.81	1.03	3.04	0.1	67.2
467	54	F	1.56	87.4	110	35.91	140	90	86	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	235	304		8.11	24	0.94	189	227	40	130	38	1.98	0.96	2.9	0.27	68.8
468	73	M	1.655	78	92	28.48	120	80	76	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	89	120			33	1.3	136	212	53	102	27	2.03	1.37	3.14	0.2	54.1
469	63	F	1.61	80	110	30.86	150	70	88	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	163	178		7.2	34	1.19	164	256	52	167	33	2.65	1.37	2.36	0.28	48.5
470	55	M	1.8	80	92.5	24.69	136	90	82	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	102	109			21	0.75	135	175	31	114	21	6	1.05	3.25	1.36	103.3
471	27	M	1.724	58.5	78	19.68	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	88	88			23	0.66	57	130	35	79	11	1.89	1.43	4.12	0.09	132.5
472	52	M	1.68	57.4	83	20.34	150	90	84	Y	N	Y	N	N	N	N	N	N	N	N	Y	Y	N	N	N	272	361		9.44	19	0.7	82	175	33	113	16	3.11	1.29	2.59	0.33	108.5
473	60	F	1.575	75.7	107	30.52	110	60	80	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	87	118			25	0.77	74	206	61	124	15	1.05	1.37	3.35	0.24	83.9
474	60	F	1.49	85	106	38.29	110	70	88	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	108	114			15	0.49	84	154	40	92	17	3.76	1.05	3.12	0.1	105.9
475	60	F	1.555	63	97	26.05	130	80	78	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	107	142			18	0.64	103	227	43	150	21	5.54	1.08	2.83	0.07	97
476	29	M	1.57	73	95	29.62	130	80	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	78	97			19	0.71	115	169	41	105	23	1.41	1.17	3.48	0.07	126.8
477	48	F	1.605	86.3	107	33.50	150	90	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	190	128		5.21	15	0.58	78	169	47	97	16	5.03	1.12	3.05	0.08	109
478	57	M	1.69	89	110	31.16	160	110	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	123	172		5.02	19	0.83	161	181	39	113	32	2.59	1.27	3.6	0.22	97.7
479	51	F	1.453	49.4	91	23.40	110	70	82	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	141	185		7.7	17	0.52	165	173	42	105	33	3.07	1.24	3.68	0.06	110.6
480	28	M	1.78	71.6	89	22.60	120	80	68	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	84	87			19	0.79	59	132	42	77	12	2.43	1.26	3.55	0.07	122.2
481	44	M	1.68	101	114	35.79	140	100	78	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	212	129		5.9	28	0.7	161	207	46	133	11	2.78	1.19	3.08	0.14	114.8
482	35	M	1.662	52.9	68	19.15	120	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	80	94			16	0.67	87	181	30	126	17	1.01	1.16	3.37	0.16	124.5
483	55	F	1.48	97.6	119	44.56	140	90	76	N	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	129	152		7	15	0.62	84	149	47	84	17	1.92	1.01	3.15	0.12	99
484	41	M	1.668	68	97	24.44	120	90	78	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	96	108			21	0.94	81	135	34	88	16	1.59	1.06	3.17	0.3	100.3
485	52	M	1.69	74	88	25.91	120	80	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	82	85			25	0.9	125	197	27	150	25	1.89	1.06	3.04	0.19	97.9
486	36	M	1.8	83	94	25.62	130	80	66	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	79	76			25	0.7	126	185	39	129	25	1.88	1.04	3.65	0.28	121.4
487	42	F	1.615	65.4	95	25.07	100	70	78	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	72	137			15	0.5	141	203	30	149	28	4.49	0.94	2.66	0.02	119.4
488	52	M	1.68	75.2	99	26.64	120	70	84	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	84	169		5.7	21	0.7	356	257	41	171	71	7.47	1.13	3.23	0.15	108.5
489	45	M	1.668	88.4	109	31.77	110	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	83	129			15	0.7	134	208	28	154	27	1.64	1.2	2.79	0.09	114
490	45	M	1.77	74.1	99	23.65	140	70	82	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	243	444		8.64	21	0.7	66	102	33	57	13	2.05	1.53	3.04	0.06	114
491	40	F	1.537	77.5	96	32.81	110	80	76	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	94	133			16	0.6	197	193	30	132	39	2.84	1.67	3.49	0.16	109.3
492	35	M	1.653	77.6	104	28.40	140	100	76	N	N	Y	N	N	N	N	Y	N	N	N	Y	N	N	Y	N	329	461		8.83	24	0.9	288	217	39	148	58	1.79	1.41	3.31	0.09	110.3
493	39	M	1.55	90.9	118	37.84	120	70	80	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	72	121			18	0.6	124	213	40	156	25	1.51	1.37	3.48	0.1	126.7
494	33	M	1.65	57.1	82	20.97	110	80	82	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N		103			16	0.7	69	187	36	136	14	1.51	1.39	3.48	0.1	124
495	61	F	1.5	56.8	97	25.24	120	80	86	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	86	113			24	0.5	120	197	36	143	24	4.02	1.25	3.45	0.22	104.5
496	68	M	1.62	66.1	98	25.19	140	80	78	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	114			7.4	17	1.2	196	150	36	91	34	3.37	1.78	2.75	0.05	61.8
497	41	F	1.49	68.1	98	25.18	120	80	82	N	Y	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	95	208		6.2	15	0.5	333	154	36	64	67	3.53	1.12	3.05	0.05	120.2
498	43	F	1.565	49.4	86	20.17	100	70	86	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	57	226		6.4	23	0.5	102	173	34	122	20	0.963	1.19	2.96	0.05	118.5
499	32	M	1.7	86.9	98	30.07	140	90	84	Y	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	107	225		7.8	24	0.5	260	197	38	111	52	1.33	1.26	3.66	0.1	143.4
500	37	M	1.63	66	83	24.84	130	90	70	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	98	133			19	0.9	99	188	40	107	20	4.05	1.11	2.98	0.06	108.7

S.No
Age.Yr
Sex
Ht(m)
Wt(Kg)
WC(cm)
BMI
SBP
DBP
PR/min
Nativemedicine (Y/N)
HT(Y/N)
DM(Y/N)
HYPOTHYROID(Y?N)
MI(Y/N)
CARDIACVALVULARILLNESS
SURGERY
RENALCALCULI
CVA
Farmer(Y/N)
Agrochemexposure(Y/N)
Nonfamer(Y/N)
Dye/Toxinexposure(Y/N)
Smoking(Y/N)
Alcohol(Y/N)
F.H.Renaldis.(Y/N)
FBS(mg/dl)
PPBS(mg/dl)
RBS(mg/dl)
HbA1C(%)
B.Urea(mg/dl)
S.Creatinine(mg/dl)
TGL(mg/dl)
TC(mg/dl)
HDLc(mg/dl)
LDLc(mg/dl)
VLDL(mg/dl)
TSH(μU/L)
FreeT_4(pg/ml)
FreeT_3(ng/ml)
UrinespotPCR
eGFR

CASE RECORD FORM

Serial No: **Hospital No OP/ IP**

Name of the Subject:

Age: **Sex:**

Address:

Location: urban/ semi urban/ rural

Height: **Weight:** **BMI:** **Waist circumference**

B.P **Pulse rate:**

Provisional Diagnosis:

Past History:

H/O any drug intake:

Present Complaints:

Personal History:Occupation; Farmer / Non farmer:

If Farmer exposure to Agro-Chemicals:

If Non farmer Exposure to dye /toxic chemicals like lead/others :

Substance Abuse:

Family History:

INVESTIGATION

TEST	RESULT
Blood Sugar Fasting (mg/dl)	
Post Prandial(mg/dl)	
Randoma(mg/dl)	
Blood Urea(mg/dl)	
Serum Creatinine(mg/dl)	
Lipid Profile Triglyceride(TGL)(mg/dl)	
Total Cholesterol (TC)(mg/dl)	
High Density Lipoprotein Cholesterol(HDLc)(mg/dl)	
Low Density Lipoprotein Cholesterol(LDLc)(mg/dl)	
VeryLow Density Lipoprotein Cholesterol(VLD)(mg/dl)	
Thyroid Profile Thyroid Stimulating Hormone(µIU/L)	
Free Thyroxine (FT4) (pg/ml)	
Free Triiodothyronine (FT3) (ng/ml)	
Spot Urine Protein Creatinine ratio (UPCR)	
Estimated Glomerular Filtration Rate (eGFR)ml/mim/1.73m^2)	

Other relevant Investigation if any:

Chennai Medical College Hospital & Research Centre
Irungalur, Trichy – 621 105.

Consent Form

You are requested to participate in a study conducted in the Department of Biochemistry, Chennai Medical College Hospital & Research Centre, Irungalur, Trichy, Tamilnadu titled "A study on eGFR, Protein –Creatinine ratio, Thyroid and Lipid profile in assessing risk factor for renal dysfunction ". Your participation in the study is voluntary

- There will be no cost for participating in the study
- Your participation is not a compulsion
- You have the right to withdraw from the study at any time.

Nature of Study:

- ✓ If any abnormalities are identified, you will be informed for further consultation.
- ✓ The results of this study will be kept confidential

We believe that the results of this study will be beneficial for advancements in medicine & Science. We assure you that we will not use these result for any other purpose.

Consent

I Mr /Mrs / Ms_____
residing at _____
_____on this day _____ after having read the consent form carrying information for the above mentioned study and I hereby give my consent to take 4ml of my blood sample for the purpose of doing serum electrolytes. I was explained about the procedure in detail and give my consent for participating in the study and for using the results for Medical & Scientific purposes.

Signature of the participant Signature of Witness
Signature of the Investigator

சென்னை மருத்துவக்கல்லூரி மருத்துவமனை மற்றும் ஆராய்ச்சி மையம்,

இருங்கலூர், திருச்சிராப்பள்ளி - 629165

ஒப்புதல் படிவம்

சென்னை மருத்துவக்கல்லூரி மருத்துவமனை மற்றும் ஆராய்ச்சி மையத்தின் உயிர் வேதியியல் துறையில் நடத்தப்படும் இ.ஜி.எப்ப்.ஆர், புரதம் - கிரேட்டினின் விகிதம், தைராய்டு மற்றும் கொழுப்பு சுயவிவரம் ஆகிவற்றினால் சிறுநீரக செயல் பிறழ்ச்சியின் ஆபத்து காரணியை மதிப்பிடும் ஒரு ஆய்வில் பங்கேற்குமாறு உங்களை கேட்டு கொள்கிறோம்.

- இப்பரிசோதனைக்கு சம்மதிப்பது உங்கள் விருப்பத்தை பொறுத்தது.
- இச்சோதனைக்கு கட்டணம் கிடையாது.
- கட்டாயம் ஏதும் இல்லை.
- பரிசோதனையில் இருந்து எந்நேரமும் விலக தங்களுக்கு முழு உரிமை உண்டு.

இந்த ஆய்வின் முடிவுகள் மருத்துவம் மற்றும் விஞ்ஞான முன்னேற்றத்திற்கு உதவும் என்று கருதுகின்றோம். இவைகளை வேறு எதற்கும் பயன்படுத்தப்பட மாட்டாது என உறுதியளிக்கிறோம்.

ஒப்புதல்

நான் திரு/ திருமதி/ செல்வி/_____
முகவரி_____
_____நாள்_____ அன்று
மேற்கண்ட ஆய்விற்காக தகவல் படிவத்தினை படித்து, கேட்டு, புரிந்துகொண்டு இந்த ஆராய்ச்சிக்கு தேவையான சோதனைக்கு என்னிடம் இருந்து 5 ml இரத்தம் எடுத்து கொள்ள அனுமதிக்கிறேன். என் மனப்பூர்வமான சம்மதத்தை அளிப்பதோடு இந்த ஆய்வின் முடிவுகளை மருத்துவம் மற்றும் விஞ்ஞான நோக்கத்திற்கு பயன்படுத்த ஒப்புதல் அளிக்கிறேன்.

பங்கேற்பாளர் கையொப்பம்

நடுநிலை சாட்சியின் கையொப்பம்

ஆய்வாளர்/ சம்மதம் பெறுபவர் கையொப்பம்

சென்னை மருத்துவக்கல்லூரி மருத்துவமனை மற்றும் ஆராய்ச்சி மையம்,

இருங்கலூர், திருச்சிராப்பள்ளி - 629165